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ECTOPIC TESTIS AS A CAUSE OF URETERAL DILATATION —CASE REPORT*

BY GEORGE C. PRATHER, M.D.†

WHILE crypt-orchidism in young adults is said to have an incidence of between .1 and .2 per cent[1], ectopic testis in the true sense of the term occurs much less frequently. Although ectopic testis is due to aberrant migration, the cause of aberrant migration is not definitely known. Sebileau and Descomps[2] have given an excellent classification of the various situations in which an ectopic testis has been reported. This is used in Young's[3] Textbook. It is as follows:

Aberrant Migrations.—

1. Intra-abdominal.

(a) Pelvic, in which the testis lies in the true bony pelvis adjacent to the posterior lateral surface of the bladder on top of and lateral to the corresponding seminal vesicle. Such a case has been reported by Charpy and DesForges-Merie[4]. In their report there is no mention of the size or course of the ureter in relation to the ductus deferens on the corresponding side. Apparently, however, the ureter was of normal size as it was confused at first with the vas. The seminal vesicles were normal in shape and position. Examination of the fluid from the vas showed no spermatozoa. From the testis apparently led the gubernaculum which followed up to the inguinal canal, through it and down to the scrotum. Microscopically this cord was composed of a number of smooth muscle fibers with abundant arterial blood vessels. It is assumed that the ectopic condition occurred in spite of the usual insertion of the gubernaculum.

(b) Deep crural, signifying a condition in which the testicle emerges through the femoral ring to the crural region.

2. Intra-abdominal.

(a) Superficial crural, in which the testicle is found in the superficial tissues of the femoral triangle.

(b) Cruroscrotal, in which the testicle is found in the cruroscrotal fold.

(c) Pubopenile, in which the testicle is located at the root of the penis.

(d) Penile, case reported by Guermonprez and Poupart[5] in which the testicle was located under the skin of the penis.

(e) Subcutaneous abdominal, the testicle lying under the skin of the anterior wall.

(f) Perineal, in which the testicle is found in the perineum.

(g) Transverse ectopia, in which the testicle is located in the opposite scrotal sac.

The case report which follows is concerned with the first division of the classification in which the testis was located in the true bony pelvis with corresponding change in the length and direction of the vas deferens, so that it constricted the ureter ½ cm. above the bladder wall. In the dissecting room I have found that normally the vas crosses the ureter about 2 cm. from the bladder. Although I have not been able to find such a complication of ectopic testis in the literature, from the manner in which the vas deferens normally crosses the anterior surface of the ureter there would seem to be no anatomical reason why such obstruction could not occur more frequently, especially with any change in direction or tension of the vas.

The patient, Mr. C. M., aged twenty-seven, x-ray technician by occupation, married and the father of one child, was seen April 5, 1934, with an immediate history of dull pain in the left flank covering a period of two weeks during which time there had been a temperature of 102° for five days not accompanied by nausea or vomiting. There has been some frequency, no nocturia and slight dysuria.

Past history: General health excellent. Four years ago an attack of left flank pain occurred which disappeared spontaneously, dull in character, not studied at that time.

Physical examination: Was normal except for the abdomen and external genitalia. Abdominal examination showed a left kidney which was enlarged and easily palpable on inspiration. At the time of examination this was not tender. The right testis was normal in size, location and consistency while the left could not be felt either in the inguinal canal, scrotum or perineum. The prostate on rectal examination was normal in size and consistency. A voided specimen of urine showed no albumin, no sugar but a microscopic sediment was loaded with pus and motile bacilli. Office cystoscopy demonstrated a marked cystitis, a normal right ure-

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teral orifice, a slightly edematous left ureteral orifice without ureterocele. Intravenous pyelography revealed the condition shown in figures 1 and 2, depicting a tremendous dilatation of the left ureter throughout its entire extent as well as dilatation of the left kidney. The right kidney and ureter were showed an appearance time of four minutes on the right, none on the left. Fifteen per cent phthalein in fifteen minutes from the right, none on the left. Further x-rays taken at that time showed no evidence of stone in the lower left ureter.

Laboratory data: Red blood count 5,310,000. Hemoglobin 94 per cent. White blood count 11,400. N.P.N. 29. Blood sugar 118. Hinton negative.

With these findings it seemed as though ureterocele, stone and congenital stricture had been defi-

FIGURE 1. Intravenous pyelogram showing dilated left kidney and upper ureter. (Dr. George C. Prather.)

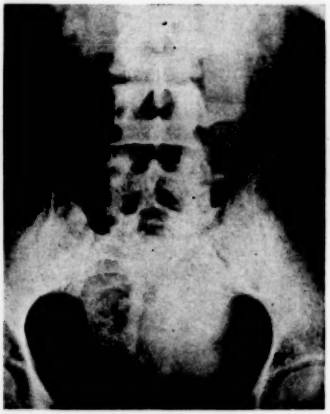

FIGURE 2. Intravenous pyelogram showing markedly dilated left lower ureter. Right kidney and ureter appear normal. (Dr. George C. Prather.)

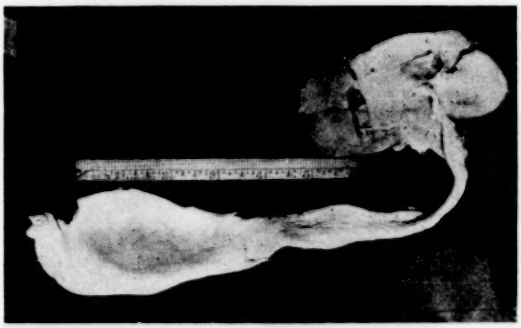

FIGURE 3. Specimen removed—ureter opened. Shrunk in fixing. (Dr. George C. Prather.)

normal in outline. Cystoscopy in the hospital April 7, 1934, confirmed previous cystoscopic findings. A No. 6 catheter entered the left ureteral orifice easily and was inserted a distance of three inches without meeting obstruction. Very cloudy, thick urine drained from this ureter. The right ureteral orifice was catheterized easily, obtaining clear urine from that kidney. Right sediment negative, left sediment loaded with w.b.c. Intravenous phthalein

nitely ruled out as the cause of the dilated left kidney and ureter. Congenital dilatation of the ureter appeared to be the most reasonable diagnosis except for a possible obstruction by the vas deferens as the result of an ectopic testis. However, with the testicle not palpable, abnormal relation between the ureter and an ectopic testis seemed the most logical diagnosis. In view of the absence of function as well as marked infection accompany-

ing the tremendous dilatation of this ureter and kidney, ureteronephrectomy was advised rather than a transplant of the dilated ureter into the bladder.

Operation, April 12, 1934.

Under spinal anaesthesia a left Gibson incision was made and the lower quadrant muscles divided. The ureter was approached extraperitoneally and found tremendously dilated to the size of the colon. It was freed and dissected down to the bladder wall where it became abruptly narrowed and was crossed on its anterior surface at this point transversely by a well-developed vas deferens the color and consistency that one usually sees in vasectomy. Palpation for the left testis revealed a small lump approximately one cm. in diameter adjacent to the posterior wall of the bladder, although the vas was not followed down to this point, nor was the testis exposed. After the vas had been retracted the ureter was clamped as it emerged from the bladder wall, bisected, stumps carbolized and tied. The ureter was then freed upward above the pelvic brim and tucked extraperitoneally into the left flank. This incision was closed in three layers with a rubber dam wick to the stump of the ureter. The patient was then turned on his side and the usual oblique kidney incision made. The muscles were divided, and the kidney identified extraperitoneally and freed. It was slightly enlarged but definitely softer than the average. The lower portion of the ureter which had been freed through the first incision was easily reached and pulled up into the incision. Ureteronephrectomy was done easily, removing the kidney and ureter as one specimen. The pedicle was clamped securely and tied. A rubber dam wick was placed to the kidney fossa and the wound closed in four layers. Figure 3.

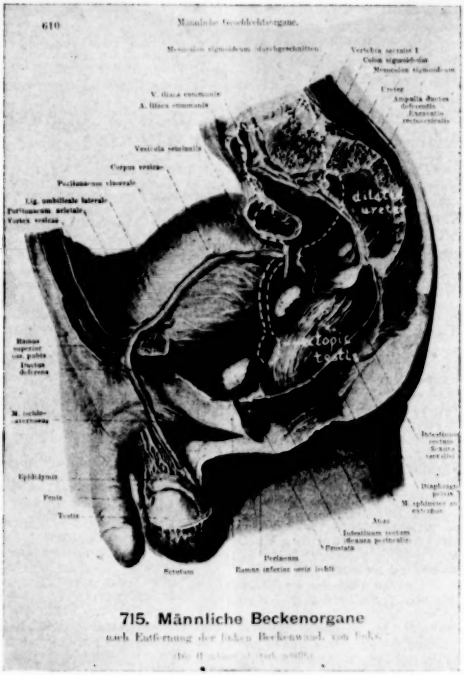

FIGURE 4. Diagrammatic change in anatomical relation between left vas deferens and left ureter. (Picture from Handatlas der Anatomie des Menschen-Spaltholz.) (Dr. George C. Prather.)

The convalescence was uneventful, both wounds healed by primary union. The patient was discharged sixteen days after operation.

Pathological report as follows: "Specimen consists of left kidney and ureter, weight 100 grams. Kidney measures 12 by 6 by 4 cms: ureter 34 cms. in length and 4 cms. in circumference at the distal end. The capsule of the kidney strips easily, leaving a smooth reddish surface. Upon opening the ureter and kidney pelvis the latter is found to be dilated as are the calices. The thickness of the remaining kidney tissue is 1.3 cms. The markings are distinct. The pelvis mucosa appears thickened, is white except for a few scattered areas of ecchy-

mosis. The mucosa of the ureter is white and slightly thickened. Microscopical examination of the ureter shows marked thickening of the wall with a diffuse lymphocytic plasma cell infiltration. Sections of the kidney show little pathology other than that associated with the chronic inflammatory process in the pelvis. The latter is characterized by lymphocytic infiltration of the wall and edema.

Diagnosis: Hydronephrosis.
Hydroureter.

This report demonstrates one of the less frequent causes of ureteral obstruction. Figure 4 represents a reconstruction of the anatomical condition, the picture from Spalteholz representing the normal relations, the dotted lines showing the condition as found at operation. It may prove interesting to observe even in cases of arrested migration in contrast to aberrant migration of the testis whether ureteral dilatation occurs with any degree of regularity. In view of the anatomical relations at the lower end of the ureter such a possibility is evident.

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ADENOCARCINOMA OF KIDNEY RECURRENT AFTER TWENTY YEARS*

BY ROGER C. GRAVES, M.D.,† AND ROY E. MABREY, M.D.†

THE recurrence of renal adenocarcinoma twenty years after an apparently successful nephrectomy for this disease, is so unusual that the following case seems worthy of brief mention.

An American metal roller, aged fifty-three, entered the Pondville Hospital in March 1933, complaining of a swelling of six months' duration, in the region of his right nephrectomy scar. His story was that twenty-two years before, he had passed bloody urine on one occasion. There were no urinary symptoms. Two years later he passed several clots of blood, and at that time developed acute retention. He was referred to a hospital for cystoscopic examination, but he was soon discharged with the report that an operation was not necessary. Three months later he again suffered from urinary retention, and after examination by Dr. Chute, he entered the Rhode Island Hospital, where a right nephrectomy was done. Following the operation the patient remained symptom-free until about six months before admission to Pondville. At that time he began to notice a swelling in the region of the scar in his right loin. At first this was not accompanied by pain, but it gradually increased in size during the next three months. He then began to complain of some pain in the right hip. There had been no urinary symptoms since the operation twenty years ago. He reported that recently he had been constipated, and that there had been some swelling of his ankles. He became tired easily and his general health had become progressively worse in the past six months.

No family history of cancer was obtained. The past history was unimportant. The patient was married and had six children, all of whom were reported to be living and well.

Examination found a well-developed and poorly nourished man of fifty-three. He was pale and gave evidence of moderate loss of weight. There was some transverse enlargement of the heart and the sounds were of poor quality. Blood pressure: 140/60. There was edema of the feet and legs.

The anterior chest and abdomen presented a striking picture of dilatation of superficial veins. The liver was markedly enlarged and irregular. The spleen was palpable and somewhat enlarged. It was thought that the left kidney could be felt. There was a small umbilical hernia. In the right flank was found the scar of the nephrectomy, and in its midportion was a slightly raised, hard, irregular mass, about 10x6 cm. It was non-tender and appeared to be fixed to the underlying structures. There was no redness or ulceration of the skin.

Rectal examination disclosed external hemorrhoids and a small benign prostate. The urinary sediment contained a few epithelial and red blood cells. The phthalein test of renal function gave an output of twenty-five per cent of the dye in the first hour, and fifteen per cent in the second. N.P.N.: 44.5 mg. Blood-sugar: 81 mg. per 100 cc. Red blood count: 3,140,000. Hemoglobin: sixty-two per cent. White blood count: 7,100. The blood Wassermann and Hinton tests were negative. X-ray examination of the skull, spine, chest, pelvis, femora, and humeri, showed no evidence of metastases. Examination of the stomach: "Fluoroscopic examination of the abdomen before administration of the barium showed a large mass centrally located, extending somewhat over to the left side, sharply outlined by the gas shadows in the colon. The colon extends upward quite high on the left side under the diaphragm and comes down to well below the umbilicus and limits the lower part of the mass. It is then lost in the shadow of the hepatic flexure which seems to be displaced downward by the large liver. After the administration of the barium the stomach shadow shows up over the shadow of the colon and is at the left and lower part of the mass. It is somewhat adherent to it. The first portion of the duodenum is displaced downward. There is no evidence of disease in the stomach itself. The gastric rugae are easily identified throughout. The first portion of the duodenum is normal in outline. The stomach seems to be emptying in almost normal time. There was no fluid retention before the administration of the barium. The loop of the duodenum is not enlarged. The mass is either the lobe of the liver or a large retroperitoneal mass. The colon shows no filling defects and there is no evidence of organic disease of the large bowel."

Biopsy examination was made of two small sections of tissue removed under novocain from the mass in the line of the scar in the right flank. The tumor was found to be highly vascular, requiring

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both ligatures and pressure for the control of bleeding. The report was as follows: "The area of the tumor in the region of the right nephrectomy scar was painted with iodine and infiltrated with novocaine. The mass was incised and two small blocks of tissue were removed for examination. There was profuse bleeding which was controlled by ligatures and pressure. A pulsation was felt in the region of the right flank, although this was not confined definitely to the tumor. It seemed to be of deeper origin. The wound was closed with silk, and a pressure dressing was applied."

Microscopic Examination: "Sections of tissue composed of dense fibrous tissue and tumor cells. Tumor cells are arranged in atypical acinar formation. They have clear foamy cytoplasm and small granular vesicula nuclei. Fairly abundant connective tissue stroma. Diagnosis: Renal adenocarcinoma. Shields Warren, M.D."

The original reports of twenty years ago were obtained and were as follows:

The patient was admitted to the Rhode Island Hospital January 13, 1913. Operation January 13, 1913. Operative Note: "Removal of right kidney. Right lumbar incision. Kidney exposed. Right kidney found enlarged and new growth infiltrating peritoneum and surrounding tissues. Renal vessels and ureter clamped and tied off with No. 2 chromic sutures. Kidney removed with considerable difficulty on account of its size. Four-fifths of kidney substance was involved by new growth. One cigarette drain inserted. Wound closed with No. 2 chromic and plain gut. Skin closed with silk."

"Microscopic Examination: Section shows specimen to consist of connective tissue stroma, in places containing cells undoubtedly derived from adrenal tissue. The cells are round or oval and there is a slight tendency toward an alveolar arrangement. There is considerable hemorrhage into the tissue."

The patient was discharged January 23, 1913, and the discharge diagnosis was hypernephroma.*

On May 21, 1933, two months after admission to the Pondville Hospital, the patient returned home to remain under observation in the outpatient clinic. The wound in the right flank was well healed. Because of the extent of the disease, neither radium nor x-ray treatments had been given.

He was next seen on June 14, and at that time his general condition seemed to have improved. He still complained of swelling of his legs. There were no urinary symptoms. On examination the tongue was moist and the pulse normal. There were no palpable lymph nodes in any region. The dilated superficial veins of the chest and abdomen, and the abdominal masses were essentially as previously described. There was no evidence of ascites. It was impossible to be certain with reference to the mass in the right upper quadrant. While it might be liver, it was felt in view of the history that it could be of retroperitoneal origin arising from the region of the renal fossa on the right side. The left kidney could not be felt definitely. Palpation could not determine accurately whether the mass to be felt on the left was of hepatic, splenic, retroperitoneal gland or renal origin. The tumor in the line of the right nephrectomy scar seemed smaller and quite freely movable. There was marked edema of the lower legs.

On October 25, 1933, the social service worker visited the patient and reported that he was no longer able to be up and about. His legs were badly swollen and he was quite weak. Frequent visits were being made by the family physician. There was no complaint of pain. A final report was received later that death occurred on January 1, 1934, and in response to a request for further information, Dr. Ralph P. Kent, of Attleboro, wrote: "It would seem almost unbelievable that the tumor which finally developed in the old scar was a real recurrence of the original growth but to the best of my knowledge and belief there was no tumor in the remaining kidney. The final clinical picture was that of general ascites, marked dilatation of superficial veins of chest and abdomen, portal obstruction, failure, of course, of the heart to meet the work."

A positive diagnosis cannot be made in this case as the evidence is insufficient; there was no postmortem examination. The clinical assumption was that we were dealing with a recurrent tumor at the site of the original operation, but metastasis from a carcinoma in the remaining kidney cannot be disproved. However, whether recurrence or reoccurrence, the reappearance of a malignant renal neoplasm twenty years after nephrectomy seemed sufficiently interesting to warrant this brief report.

*For some time there was a tendency among pathologists to designate as hypernephromata tumors with vacuolated cells suggesting an adrenal cell origin. Dr. Warren feels, with regard to this case, that in the absence of direct chemical determination of the material within the vacuoles, it would be difficult to prove adrenal origin, and that in view of the later slides these were probably vacuolated renal cells.

KERATODERMIA BLENNORRHAGICUM*

(Gonorrheal Dermatitis)

BY AUGUSTUS RILEY, M.D.†

I WISH to show some lantern slides that demonstrate skin lesions, which are more or less a curiosity because of the rarity of such lesions, and the controversy among some dermatologists, and between dermatologists and internists as to the exact cause of these lesions.

There have been only three cases seen and diagnosed as keratodermia blennorrhagicum in the Boston City Hospital. The first case entered the hospital about three years ago. He was examined and studied by Dr. Downing, in the Dermatological Department. The case with a review of the literature was reported by Downing[1] who states that the lesions, he believes, occur more often than they are correctly diagnosed. He feels that the lesions in many instances resemble the lesions of psoriasis so closely that it is hard to differentiate the two. As the

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FIG. 1. Roughened and crusted areas on palm of hand.

In addition to the skin lesions as shown here by the lantern slides, these three cases had swollen, painful and tender joints.

There is a history of a gonorrheal urethritis with inadequate or no treatment. Prostatic and vesicular secretion obtained by massage of these parts shows much pus. Downing believes that this disease is due entirely to a gonococcal infection, and that the site of this infection is in the prostate and vesicles. Therefore, the best treatment for these cases is surgical eradication of the site, that is vesiculectomy and prostatectomy.

Two of these cases have been operated on by me and in both complete vesiculectomies were done, with incision and drainage of their prostates. The results were complete relief of their joint symptoms after short intervals and a complete disappearance of the skin lesions after a much longer time.

The third case is now being studied to determine if possible whether the lesions are due to the invasion of the gonococci into the tissues, or the results of some toxins.

These lantern slides were made of the first case operated on, and they show very clearly the

FIG. 2. Piled up crusts between the toes and at the base of the toe nails.

FIG. 3. Destructive lesions on soles of feet.

lesions progress there is a more destructive process to the skin than in psoriasis, and there is a "piling up" of crusts covering the lesions to a much more marked degree than one expects to find in psoriasis. When the lesions begin between the toes it is more likely to be called "Athlete's Foot".

"piling up" of the crust and a destruction of the skin of the soles of the feet. This case had extensive lesions of the whole back, extending from the shoulders down over the buttocks.

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CALCIFIED HYDROCELE OF THE TUNICA VAGINALIS TESTIS*

Case Report

BY C. J. E. KICKHAM, M.D.†

TWO years ago, it was my privilege to report before this Association a case of "Calcified Hydrocele Simulating Tumor." At the Pondville Hospital recently, we encountered a patient in whom a similar pathological entity was present. However, the calcified process in this case was found to be far more advanced than that in the case previously reported.

X-ray of the external genitals and pubic bones delineating calcification of a hydrocele in the right scrotum.

The patient, Pondville Hospital Number 7712, a retired bookkeeper of eighty-six years, was admitted on April 10, 1933 with a complaint of bleeding from the rectum of two months' duration. For eight months previous to admission, he experienced considerable rectal tenesmus, diarrhea, and inability to control his bowels. These symptoms had become progressively worse. He stated that there had been a mass present in the right side of his scrotum for over forty years. As far as he could tell, however, there had been no appreciable increase in the size of the mass over that long period. Also, there had been no pain or discomfort associated with the condition. There was no history of injury.

Examination revealed the presence of a stony, hard, non-tender mass, somewhat larger than a hen's egg, in the right scrotum. It did not transmit light. Palpation of the mass elicited a deep grating crepitation. It was not subcutaneous. The spermatic cord was normal. The epididymis could not be identified. The scrotal skin was not adherent. The left scrotal contents were normal. Rectal examination disclosed an advanced inoperable malignancy of the rectum. A calcified hydrocele seemed to be the most probable diagnosis. However, the remote possibility of calculi was considered in view of the grating crepitation which suggested the presence of a "bag of stones". The Prolan A test to determine the presence or absence of anterior pituitary hormone in the urine was not carried out as the history and clinical findings excluded the diagnosis of neoplasm of the testis.

X-ray examination of the external genital region delineated a circular area of calcification, measuring 5 cm. in diameter in the region of the right testis, consistent with calcification of a hydrocele.

In view of the patient's age and the presence of advanced inoperable malignancy, no specific treatment of the condition was indicated. A program of general care and symptomatic treatment was carried out and the patient expired on October 13, 1934. Permission for postmortem examination was obtained.

Pathological report of the specimen: the contents of the right scrotum removed. Specimen consists of a calcified sac 6x5x5 cm. The wall is thickened and measures .2 to .5 cm. On opening the sac, one finds about 15 cc. of milky fluid with distended particles having a distinct glistening sheen. The inner wall of the tunica is covered with ragged brownish putty-like material with an underlying calcified bed. The testis is encased by this thickened tunica and is compressed posteriorly. The epididymis does not appear remarkable. Diagnosis: Calcification of a hydrocele of the tunica vaginalis testis.

The above case is reported in view of the unusual clinical and pathological findings. It emphasizes the value of x-ray examination in masses of the scrotum which do not transmit light and the importance of crepitation as a diagnostic sign. The Prolan A test should be carried out in all cases of scrotal enlargement in which the diagnosis of malignancy has not been excluded.

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REPORT OF UNUSUALLY LARGE MALIGNANT GROWTH IN UNDESCENDED TESTIS*

BY EDWARD J. O'BRIEN, M.D.†

McKENZIE and Ratner in a recent communication reviewed the literature on this subject and at the same time reported three new cases. In 1931 they made a report on tumors of the testis. In that report they stated that they did not believe undescended testes per se were seriously predisposed to malignant degeneration, and according to their most recent offering in which they cover the literature quite thoroughly, they have not changed their opinion. In reviewing the literature on the subject there is a wide variation in reports. Various authors differ widely in their figures, so much so that some reports asserted that tumor formation occurs anywhere from 30 to 300 times as often in the undescended as in the normally placed testicle. There is also a vast difference of opinion as regards the relative infrequency in inguinal and abdominal cryptorchids. McKenzie stated in his 1931 communication that he had not seen a case of malignancy in a series of 105 cases of undescended testis but since that time has had thirteen additional cases of tumor of the testis in which three were in undescended testes. Some of the contrasts in the reports I will cite:

Sixty-seven cases of tumor of the testis were reported by Cunningham, none of which were in an undescended testicle. Kocher found only one case of malignancy in 1000 cases. Eccles reports 859 cases of undescended testis without finding malignancy in any of them. In a series of 1357 cases Coley did not have a single case. McKenzie further states that in looking over the medical records of the World War there have been three and one tenth cases to every 1000 men examined and he uses this as a basis for his theory that undescended testes are not predisposed to malignancy. He feels that there should have been many more cases reported but no mention is made of them. Other authors vary in their reports, for Hinman reviewed 649 cases of cancer of the testis and found twelve and two tenths per cent in undescended testicles.

Regarding the treatment of this condition there is also some difference of opinion. Some authors who are firmly convinced of the potential malignancy of the undescended testis advise orchidectomy in every case. And to emphasize their point they cite the case of Dr. Fred Lund who in 1924 reported a case that developed malignancy after orchidopexy. In his search of literature McKenzie states that he was able to find only one similar case and that was reported by Rea.

J. H. W. aged thirty-nine, male, single, was admitted to the Somerville Hospital on November 20, 1933.

Family History: Mother died of arteriosclerosis in 1929. Father living and well. One brother and three sisters living and well. One brother and one sister died in childhood.

Past History: Scarlet fever. Occasional cold. Duodenal ulcer for five years. Auto accident with scalp wound. No operations.

Present History: For the past year has had symptoms referable to lower abdomen and pelvis. Periods of constipation. Frequency and occasional dysuria. Irregular pain in left lower abdomen radiating to the left back and left thigh. Appetite good except when ulcer was active. Sleeps well. Best weight 190 lbs. Lost 20 lbs. in past year with diminished "pep".

Physical Examination: Well-developed and nourished young man. Pulse, temperature and respirations normal on admission.

Head: Skull symmetrical, no exostoses or injuries. Scalp clean; no scars. Hair of good growth and texture.

Skin: Color normal, clean, warm and moist and of elastic texture. No purpuric spots, rashes or other lesions.

Eyes: Pupils equal, regular and react to light and accommodation. No lid-lag, strabismus or nystagmus. The conjunctivae show no hemorrhage, jaundice or injection.

Ears: Hearing good, no tenderness or discharge.

Nose: No discharge, deformity or obstruction. No tenderness of sinuses.

Mouth: Mucous membranes of good color. Teeth well preserved. Tongue protrudes in the midline with no tremor. Tonsils are neither hypertrophied nor injected. Pharynx shows no postnasal discharge or injection.

Neck: No abnormal pulsations. Thyroid not palpable.

Chest: Symmetrical, expansion equal on both sides. Heart: Apex impulse is in the fifth interspace within the nipple line. No palpable thrill, percussion reveals no enlargement or displacement. Heart sounds are regular, not rapid, and of good quality. No murmurs heard. The pulse is regular and of good quality. Lungs: No change in tactile or vocal fremitus. Breath sounds are of the normal character. No râles and no friction rubs.

Abdomen: Not distended but presents a low abdominal tumor which is hard, irregular, not especially tender and about the size of a five months' pregnancy.

Genitals: Normal except that the left testicle is not in canal or scrotum. Inguinal ring on left side admits finger tip.

Extremities: No deformities, paralyses, abnormal sensations, tremors or atrophy. No varicosities or edema.

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Reflexes: Equal and active. No Babinski or ankle clonus.
Rectal examination: Negative.

Short report of G. I. series by Dr. Paul Butler: The examination of the gastro-intestinal tract showed a deformity in the first part of the duodenum, which is quite typical of a duodenal ulcer. There was some pressure on the lower small intestine and on the sigmoid flexure. This is evidently caused

Photograph of unusually large malignant growth in an undescended testicle.

by a large tumor mass in the lower abdomen. This mass apparently does not involve the bowel and is not causing any obstruction.

Laboratory data:
Urine: straw-colored, clear, 1020, acid. Albumin, none. Sugar, none. Phthalein normal. R.B.C. 4,750,000. W.B.C. 7800. Hemoglobin 70. Blood:—Nonprotein nitrogen 26 mg. per 100 cc. of blood. Wassermann-Kahn negative.

Patient was seen by me in consultation with Dr. Eugene Maguire on November 21, 1933 at which time I did a cystoscopy and pyelograms. Bladder was normal. Pyelography showed a mild degree of dilatation of the pelvis of both kidneys, and a slight kink of the upper portion of the left ureter was demonstrated. There was a shadow in the x-ray plate extending from the pelvis almost up to the lower pole of the left kidney. This appeared to be a neoplasm. Following this examination I advised laparotomy for the removal of the tumor. It is my opinion at this time that this man had a neoplasm and probably an intra-abdominal teratoma of the testicle.

November 24, 1933. Operation: Laparotomy. Low abdominal incision extending from about one inch above the umbilicus and to a point five inches below was made. On opening the abdomen a smooth lobulated tumor was seen. This filled the entire pelvis and was adherent to both large and small bowels and all the contiguous tissues, showing that there must have been considerable inflammatory reaction going on. The bladder was dissected free from the growth. With much difficulty the tumor was freed from the pelvis and it was found that the mass had a pedicle which originated from the spermatic cord. The tumor was removed and all raw surfaces covered with peritoneum. A small cigarette drain was placed in the tumor cavity to take care of any oozing. The abdominal wall was sewed up tight in layers about the small drain. The patient had an uneventful convalescence.

Pathological Examination by Dr. Timothy Leary:
Gross examination: The tumor is roughly spherical, nodular, measures 15.5 x 15 x 13 cm. in its greatest dimensions. Attached to the surface at one point are bands of fibrous tissue among which some fat is present. The spermatic cord could not be identified grossly. On section the tumor is yellowish white, translucent, homogeneous.

Microscopic Examination: Tumor is made up of alveolar masses of large cells, rounded or polygonal, with poorly staining cytoplasm and large, round vesicular nuclei-mitotic figures are fairly abundant. Tumor is fairly well encapsulated. Sections were taken from band-like adhesions on surface thought to be spermatic cord. One of these shows areolar and fatty connective tissue supporting a group of large vessels, and also smaller vessels about some of which bundles of unstriped muscle occur. In one of the smaller vessels a few tumor cells are seen, one in mitosis.

Diagnosis: Embryonal carcinoma with characteristics of testicular embryoma.

Following discharge from the hospital, the patient was closely watched and now, one year following operation, he is apparently well.

REPORT OF UROLOGICAL CASE DISCOVERED IN COURSE OF EXAMINATION FOR OTHER AILMENT*

BY EDWARD H. TROWBRIDGE, M.D.†

MR. W. K. H., aged twenty-one, weight 198¼ lbs., was treated August 13, 1934, for pustule of callous area on sole of right foot. Physical examination did not reveal any special focus of infection other than the foot, which was duly treated and the patient recovered. On the second visit, by request, a specimen of twelve hour urine, was furnished which did not disclose the condition of the kidneys at that time.

A short time after the patient had recovered, the father wished to take out a life insurance for the young man, and his urine was sent to the medical examiner of the insurance company and the young

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man was passed. The medical examiner, at the home office of the insurance company, requested a sample of urine, according to routine custom, and on examination, found pus in the urine and the young man was temporarily denied insurance with the statement, however, from the insurance company's examiner that it was probably something minor and would probably disappear.

This information was given when the young man appeared a second time at my office on October 5, 1934, when there was another small accumulation of pus in the neighborhood of the former pustule. Three specimens of urine were examined in the course of a week, and in each specimen there were detected pus, phosphatic crystals, no red blood cells, no casts, no special accumulation of epithelia suggesting the source of the pus; no hematorrhea at any time. The patient was advised to have a plain x-ray plate of the kidney, which was taken on October 22, and no shadow revealed in either kidney. On October 23, the patient was subjected to intravenous injection of hippuran with the following report from the roentgenologist: "The first radiograph made immediately after the injection of hippuran shows the pelvis and calices of the left kidney well filled. This appears to be a normal kidney with the true pelvis unusually large and the calices comparatively small. The upper calyx is much longer than the other calices, which is not unusual.

"The region of the right kidney shows no pelvis shadow, but there are indefinite shadows in the kidney itself.

"Radiographs made fifteen, thirty-five and sixty minutes after injection show no marked change in the left urinary tract. The left ureter contains a small amount of injection, with no dilatation, and the previous mentioned opaque bodies in the bony pelvis are apparently not in the ureter.

"The right kidney shows progressive filling of the calices so that finally the kidney appears to consist principally of globular masses of opaque material varying in size from ½ to 1½ inches in diameter. No injection is seen in the right ureter.

"The bladder shows nothing remarkable.

"X-ray Diagnosis: Right pyelonephritis."

On October 24, the patient was subjected to a cystoscopy, which revealed a golf-hole appearance of the right ureteral orifice, the border of which was distinctly reddened. The right kidney was duly catheterized and also the left, with an immediate appearance of urine dribbling through the left catheter, while none was discharged from the right. Phenolsulphonephthalein test was then administered and came through the left kidney in about six minutes, and very faintly a short time after from the right kidney. The left kidney secreted freely while only about two-thirds of a teaspoonful of urine was secreted from the right kidney.

The patient showed a slight reaction from the catheterization, being confined to bed for two days. Inasmuch as so little kidney substance remains in the right kidney and its function is practically nil, the parents have been advised to have the right kidney removed as early as possible.

The urine submitted to me and to the insurance physician who passed Mr. H., must have been secreted from the left kidney.

TESTICULAR BIOLOGY, SCROTAL FUNCTION AND THE MALE SEX HORMONE*

BY CARL R. MOORE, PH.D.†

DURING the last fifteen years the testis has been studied in my laboratory from the purely biologic standpoint rather than from the clinical aspects and I propose to-night to discuss with you some of the problems that suggest themselves as being of some probable interest to a group such as you represent. The first portion will deal with some of the better known phases relating to spermatogenetic activity and the second with more recent studies on hormone secretion and function.

CRYPTORCHIDISM

It is well known that both man and mammals are subject to failure of testicular descent (cryptorchidism), and perhaps less well appreciated that bilateral cryptorchidism results in sterility. Several years ago, a good deal of time was devoted to an experimental investigation of the problem of cryptorchidism.

Experimental cryptorchidism has been studied in my laboratory on rats, guinea pigs, dogs, rabbits and sheep. The operation of removing the testicle from the scrotum into the abdomen is simple, of course, in those animals where the inguinal canal remains open. For this purpose the canal is merely closed and the testicle not otherwise fastened in the abdomen. Such abdominally confined testicles, undergo a remarkably rapid degeneration of the germinal epithelium, exhibiting a complete disorganization of the seminiferous tubules within the period of one week. Fragmentation of the loosened cells, cytolysis and absorption continue so rapidly as to practically free the testicle of all germ cells within three weeks. There follows a continued involution of the tubules, containing now only Sertoli cells, and no recovery occurs however long the organ remains in the abdomen.

To answer the question whether recovery was possible after such marked degeneration, testicles in various stages of injury were replaced in the scrotum. Recoveries to the normal state occurred after apparently hopeless degeneration, and within a period of two to three months spermatozoa were again present.

Experimental cryptorchidism prior to the time of appearance of spermatozoa, such as fifteen to twenty days after birth in the guinea pig, is the nearest possible counterpart in these animals to congenital cryptorchidism in man. Such testicles returned to the scrotum five months

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later recover their spermatogenetic function and possess spermatozoa from seventy to ninety days later. Such an operation for scrotal replacement of abdominal testes of a guinea pig is roughly equivalent to scrotal replacement in a man twenty to twenty-five years old[1].

One can scarcely deal with conditions that induce degeneration so quickly and so completely without making inquiry into the cause of the injury. In brief, successful elimination of such possible causes as interference with blood supply, nervous connections, pressure, or hormonal influences centered our attention on higher than normal temperature as a possible cause for the degenerative changes.

Experiments to test such an hypothesis were begun and a brief presentation of a few of them may be of interest. We measured the temperature simultaneously in the abdomen and scrotum on the same animal and found abdominal temperatures of rats, rabbits and guinea pigs to be from one and one-half to seven degrees C. higher than those in the scrotum[2]. Harrenstein[3] (Amsterdam) found comparable differences in abdominal and scrotal temperatures in boys, the scrotum being one to three degrees cooler than the interior of the abdomen. It is thus a fact that abdominal temperatures are higher than those of the scrotum but it remained to show that the testicle was affected by such slight differences, or rise in environmental temperatures.

Unoperated guinea pigs received short treatments of increased temperatures applied to the scrotum by means of hot pads, electric heat, and water warmed from five to seven degrees C. above normal body temperature. One period of twenty minutes of application to the scrotum of temperatures a few degrees higher than body temperatures was followed within a few days to two weeks by severe testicular injury and in more severe cases by temporary or permanent sterility, depending upon the degree of exposure to such elevated temperatures[4].

Since, therefore, scrotal temperatures are lower than abdominal temperatures and since the testes are injured by short exposures to temperatures above their normal environment, and are caused to degenerate by elevation into the abdomen, it follows that the scrotum functions to lower the environmental temperature of the testicle and hence is a thermoregulator. It should follow, therefore, that interference with this thermoregulatory function should induce testicular injury even though the testicles remain in the scrotum.

Sheep present a large pendant scrotum that can be easily insulated against loss of heat. The scrotum of a ram was, therefore, loosely encased in woolen wrappings, and an outer waterproofed covering, and the animal permitted to run at large for a period of eighty days. Removal of the testicles showed the entire absence of any normal seminiferous tubules; no tubules possessed spermatozoa and the majority showed severe degeneration with many possessing only Sertoli cells. The animal had sterilized itself with its own body temperature simply because of interference with the scrotal heat-regulatory mechanism. The testicle, though in the scrotum, came under the influence of temperatures slightly above normal scrotal temperatures due to the insulating cover[5].

We can visualize, therefore, that the scrotum, thin-walled, devoid of subcutaneous fat, richly provided with sweat glands and capable of great relaxation and contraction presents a functional adaptation to a delicate need. Cunningham, a few years ago, stated "various causes have been suggested for the formation of the scrotum, but no one has ever been able to suggest a use for it".[6] It appears now that we are able to assign to it a useful purpose, that of regulating the environmental temperature of the testis. That such a function is not only useful but absolutely essential for germ cell formation is bountifully shown by the conditions in cryptorchidism.

VASECTOMY

The question of the efficacy of ligation or section of the vas deferens for the purpose of rejuvenation recurs time and again. The great impetus was given to it by the glowing accounts of Steinach of Vienna, in 1920. You will, no doubt, recall the chief contentions (1) that blocking off the excurrent ducts from the testicle induces destruction of the germinal epithelium and an increase in the interstitial tissue, (2) that increased interstitial tissue means more hormone secretion, and (3) that excessive hormone secretion leads to a real rejuvenation such that in Steinach's cases old decrepit males became sleek looking, vigorous and pugnacious youngsters. Promptly from this, and on the assumption that what is good for a rat is good for man, the operation was applied to man. The most glowing accounts of beneficial effects have been written.

Briefly analyzed we find that closure of the outlet passages from the testes of rats, rabbits, guinea pigs, dogs and sheep, does not lead to loss of all germinal epithelium and, in fact, more than 100 years ago Sir Astley Cooper in England showed that experimental ligation on the dog of five years' duration did not lead to loss of gametogenetic function. Secondly, the evidence does not support the contention that an hypertrophy of interstitial cells follows. Thirdly, if interstitial cell hypertrophy really occurred, as postulated, it cannot be taken for granted that greater hormone secretion would follow. Fourthly, it is by no means established that excessive amounts of hormone are beneficial or that testis hormone in any manner leads to rejuvenation. Three years ago a fortu-

nate case of congenital absence of all outlet passage from the testicle (absence of entire epididymis, vas, and seminal vesicles) revealed that in such an adult guinea pig the testis was normal in size, actively producing germ cells (which were gradually destroyed and resorbed) and that no interstitial cell hypertrophy had occurred. Closure of the vas, perhaps better resection, is, of course, a simple and efficacious sterilization operation but there is no acceptable evidence that it has any value whatsoever as a measure effecting rejuvenation[7].

TRANSPLANTATION OF TESTIS GRAFTS

Successful transplantation of testis tissue is a little difficult. The factors concerned are (1) obtaining successful vascular invasion of the transplant prior to necrosis of the tissue which involves relatively small amounts of tissue, (2) overcoming the normal host resistance to implants which may be cellular (lymphocytosis, phagocytosis) or serological and (3) utilization of compatible tissue which appears to be tissue from the same species.

The transplant can be placed at any point where vascularization is obtainable, subcutaneous, intraperitoneal, or within organ substance (kidney), but the scrotum has, so far, proved to be the only location in which spermatozoa develop. From well over 100 transplants from young rat donors under the best conditions available, I obtained successful incorporation of approximately fifty per cent[8]. The persistence of a palpable nodule is no evidence of graft persistence since I have sectioned quantities of such nodules only to find connective tissue or scar tissue. Viable grafts will secrete hormone but all evidence indicates that autolyzing masses of testicles do not liberate detectable amounts of hormone. With sufficiently reliable and sensitive hormone indicators to detect the secretion produced within two days by normal testes, we have been unable to detect any hormone liberation from two rat testes undergoing autolysis in subcutaneous positions[9].

INTERNAL SECRETIONS

The principal cause of the tardiness in obtaining the active principle of the internal secretions of the testis has been the lack of practical methods of identification of the substance or substances. I propose to discuss this subject with you and to lay down what appear to me to be some general principles of hormone secretion and function.

Within the last few years the application of regression of the cock's comb following castration, and its growth response to testicular hormone injections, has been perhaps the most widely used test employed. By utilization of this test, McGee in Koch's laboratory of biochemistry at the University of Chicago first successfully prepared potent hormone preparations. The active principle was obtained from the lipoid fraction of fresh testicle tissue from the bull. It was found capable of inducing the growth of the comb of capons to essentially the normal cock type.

In coöperation with McGee, Koch and Gallagher and a group of my own students we were able to develop satisfactory methods on mammals for testing for the presence of the substance and to study its biological effects. I may say that the hormone effective in the capon was also effective in mammals. The material is sex specific but is not species specific.

The *spermatozoan motility test* for the internal secretions of the testis rests upon the fact that spermatozoa in the epididymis of a guinea pig remain alive and capable of exhibiting motility when suspended in physiological saline solution for a longer period of time in the presence of hormone than in its absence. Spermatozoa confined in an epididymis surgically isolated from the testicle remain capable of motility for a period of sixty-five to seventy days if the opposite testicle is present to provide a source of hormone. When no hormone is present, capacity for motility is lost by twenty-three days after operation. Injection of testis hormone into animals just castrated permits spermatozoa to remain viable for normal lengths of time[11].

The *electrical ejaculation test* conducted upon the guinea pig depends upon inducing an ejaculation when the animal receives an electrical shock through the brain. A normal male produces an ejaculate once each week of approximately one to three grams, but a castrated male is unable to void an ejaculate because of loss of secretory function on the part of the prostate gland and seminal vesicles which are the main sources of the semen. Males castrated for six months, however, have produced typical coagulated ejaculates after ten days of treatment with testis hormone[12].

Morphological indicators of testis hormone in the rat are provided by changes occurring after castration in the prostate gland, seminal vesicles, Cowper's gland and the vas deferens. Within forty-eight hours after castration the seminal vesicles reveal changes visible in the finer cellular character of this organ and within four days in the prostate gland. Twenty days of castration bring on perfectly definite histological changes in all these structures.

The injection of testis extracts has prevented the development of all castration changes so far noted in this animal. When the injections were delayed until after such changes had occurred, complete restoration to normal morphological states has been attained by daily injections of from ten to twenty days[13].

PRINCIPLES OF SECRETION AND FUNCTION

Source: The hormone has been obtained from extraction of the fresh testicles of the bull, goat, ram and pig. It has been recovered from the urine of men but not of boys under ten years[14]. It has been found in small amounts in the blood of bulls and goats. The yield on extraction is surprisingly small and tons of bull testes as well as hundreds of gallons of human urine have been extracted in Koch's laboratory, yet insufficient amounts for laboratory investigations have been prepared. *Secretion* of the hormone is a continuous process when once initiated in the rat, and presumably in man, for in the rat a forty-eight hour period of castration is registered by morphological changes. Hormone is not stored in the body but is excreted in the urine. The majority of vertebrate animals secrete it but once a year for short periods. Though the testicles are intact, the accessory organs of reproduction are in a castrate condition for the greater part of the year. Injections of hormone restore the functional state within a week so that it is certain that the testicle does not produce the hormone in the seasonal breeding types except during the breeding season[15].

RELATION TO HYPOPHYSIS

The demonstration is now complete that regulation of testicular activity, both spermatogenetic and hormonal, does not reside in the organ itself but comes from the outside. The hypophysis or pituitary gland is at least a major factor in such regulation but it is unknown whether, or to what extent, other organs of internal secretion participate.

The brilliant work of Philip Smith and his colleague, Engle, in this country and of Zondek and Aschheim in Germany, with the many others who have participated, has revealed the dependence of the gonads upon the hypophysis and the responses of the gonads to stimulating agents[16]. Removal of the hypophysis, as well as certain hypophysial disorders leads to the inability of the gonads to produce germ cells or hormone. Introduction of hypophysial material or extracts stimulates the gonads excessively. The testis responds in a remarkable fashion with increased hormone secretion but stimulation of the spermatogenetic function is less clearly indicated. The hypophysis, therefore, produces a hormonal substance that stimulates testicular activity and the amount of testis hormone secreted is an expression of the amount of hypophysial secretion that reaches the testis through the blood stream. This amount, however, is probably always insufficient to enable the testicle to function to its full capacity, for introduction of greater quantities of the material induces greater hormone secretion as the increase in size of the accessory reproductive organs indicates.

Therapeutic value—Testis hormone function. When one raises the question of the function of the male sex hormone some answers stand out clearly, but of certain phases we are very ignorant. First, it is clear that the function relates primarily to reproduction. When spermatozoa are produced they must be so transmitted as to enable a meeting with the egg. In lower forms, particularly invertebrate, there is no evidence of the existence of testis hormone. With the vertebrates, however, the character of the genital system is more complex, consisting in mammals of the epididymis, vas deferens, prostate and Cowper's gland, seminal vesicles and penis, all of which are fundamentally concerned with providing a fluid medium for transfer of ripe spermatozoa. Out of the breeding season these structures are non-functional. A few mammals such as the rat, rabbit, guinea pig, and man remain in a continuous reproductive state, and hormone is secreted throughout the reproductive period. It is clear that all the accessory reproductive organs are controlled by testis hormone. Secondly, it is clear that the sex drive of lower vertebrates is conditioned by testis hormone. Mating responses occur only during the breeding season when hormone is being secreted: it can occur whether germ cells are produced or not, and it does not occur if the testicles have been removed. Thirdly, it is also clear that certain more or less incidental, but unimportant characters for breeding purposes are conditioned by testis hormone, such as certain horny growths of mammals, the large comb and wattles of birds, the voice of man; perhaps also certain regulations of hair growth, though this relation is by no means clear.

But to this general biological horizon of the male hormone have been added the popular conceptions of traditions and beliefs of antiquity and the unwarranted postulates of doctors of medicine. Thus the almost universal popular conception that a castrated man is of no earthly use should be contrasted with the centuries old custom of castration of domestic animals in many cases to improve them for certain purposes. Certain doctors of medicine have had a wide influence by emphasizing that certain procedures, supposedly involving manipulation of the hormone level as by testis transplantation, vasoligation, and more recently from injection of hormone, lead to increased length of life, better appetites, sharpened mentalities, sound sleep, and that many organic ailments have been benefited, such as diabetes, tuberculosis, acne, locomotor ataxia, otosclerosis, heart failure, etc.

When we examine the situation more closely for the real truth of the situation, so far as known, we find surprisingly little evidence of

a reliable character that the testis hormone in man exerts any beneficial effect aside from its control over the accessory reproductive organs; even here circumscribed data are all too few.

If we turn to the eunuch for what little information we have of a scientifically recorded nature, we find that despite the economic, social and hygienic level he is privileged to occupy, history records major attainments for the eunuch in governmental, military and general pursuits. Instead of being short-lived he is reported to be long-lived; instead of the elimination of all sexual inclination, we have reports of considerable promiscuity. Recently McCartney[17] examined twenty eunuchs and reported ten cases of gonorrhea. We have all too few records of cases such as that reported by Rowe and Lawrence in this city[18]. The energetic young man of wide travel experience had both testes removed because of certain pathology. Nine years later a successful marriage changed the psychological horizon and from a lethargic, unambitious state there emerged a revivified individual who organized a new business venture in another part of the country.

When we have developed a scientific attitude and have freed our minds from mythological traditions, we will develop a proper state for a real examination of the question of the value of male hormone to man.

One negative value appears to stand out very suggestively and that is that the hormones produced by the gonads are not gonadal stimulants. We do not know of any case in endocrinological study where the secretions of an endocrine gland exert a direct influence upon the gland secreting them. Thus insulin does not stimulate a pancreas, thyroxin a thyroid, pituitrin the pituitary, adrenalin or cortin the adrenal. Why then should we expect testis hormone to stimulate the testis or theelin or estrin the ovary? Indeed the facts show that rather than stimulation the reverse condition obtains; gonad hormones have proved injurious to the gonads. Thus injections of estrin or theelin into normal rats, dogs and monkeys lead to ovarian degeneration[19]. We have likewise shown that injections of replacement doses of testis hormone daily into normal young male rats for a period of two to three weeks are injurious to the testicles[20]. Testicular weight of such treated males compared with littermate controls is from twenty to fifty per cent reduced; tubules show injury and no spermatozoa are present. There is no information at hand that even suggests that gonad hormone would improve a hypofunctioning gonad, but every day in this country probably sees the injection for such purposes—especially theelin into women for alleged hypofunctioning ovaries.

REGULATION OF HORMONE SECRETION

An array of puzzling facts can be assembled to introduce the question of hormone secretion.

1. The testicle though capable of secreting hormone long before puberty does not secrete detectable amounts.
2. The testicle probably never exerts its maximal potentialities for hormone secretion.
3. In seasonal breeders though the testicles are present during twelve months they secrete hormone for only two, three or four months.
4. Excessive secretions are injurious to the gonads.

We have offered a working hypothesis to account for the regulation of hormone secretion based upon a reciprocal interaction between the gonad and hypophysis. Hypophysial secretions stimulate the gonads and the latter produce germ cells and gonad hormone. The gonad hormone stimulates the end organs (accessory reproductive equipment) but in sufficiently high doses lowers the secretory output of the hypophysis. This diminution of hypophysial secretion lowers the gonad hormone output and thus the oscillation of these secretions, probably also involving other secretions, is the balancing mechanism of control within the organism[21].

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DISCUSSION

DR. GEORGE G. SMITH: We have been listening to a very interesting and fascinating talk. There are few of us who can contribute a great deal to this discussion but I am sure a great many of us have questions we would like to ask Professor Moore.

There are two aspects that are of direct interest to us, that I wish Professor Moore would comment on if he cares to. One is the effect of this hormonal secretion upon hypertrophy of the prostate, whether it does influence it or not; and the other is the relation of this to the descent of the testes. I would like to know whether he thinks there is any reason to believe that descent of the testicle can be influenced by the injection of hormone of any sort.

DR. FULLER ALBRIGHT: I would like to have Professor Moore's opinion as to the best test to use in determining whether the normal male is putting out a normal amount of hormone. I would also like to ask him what estrin does to all these various tests in the castrated male, whether it has been shown that the male and female sex hormone are absolutely different. There is some question as to whether we find estrin in males and I would like to have a discussion of the difference between the male and the female.

DR. E. L. MERRITT: I was interested in what Professor Moore said about injecting theelin into rats and wrecking the gonads. Does he believe that in the case of young female children where we might use theelin, that we should endeavor to change the epithelium in treating gonorrheal vaginitis? Would there be any possibility that we might be causing some damage to the ovaries of these young female children who are not developed as yet?

DR. FLETCHER H. COLBY: I would also like to ask Doctor Moore if he could possibly apply his knowledge to some of our own particular problems. I think we all agree that testicular extracts, etc., are of no value. It evidently has to be done through the stimulation not of the testicles themselves but of the pituitary. Are there any substances which will stimulate the pituitary with a possible chance of stimulating spermatogenesis? Even if there is yet no experimental evidence of that, are we entirely wrong in employing certain preparations which we have used on the theory that there is a possibility of stimulating the pituitary and in that way possibly raising the male's lowered fertility?

DR. CARL R. MOORE: Regarding prostatic hypertrophy I do not think it is a male hormone problem but I am not certain. One can produce enlarged prostates by injecting male hormone. We have injected normal young male rats with the male hormone and obtained decreases in weight of, and injury to, the tubules of the testis. At the same time, however, the prostate may be increased four to five times that of the untreated control and the seminal vesicles even larger in proportion. I remember the figures in one case in which testis weight was reduced below the untreated control by sixty per cent, yet the seminal vesicles were increased by 800 per cent. The end organs (here seminal vesicles and prostate) will respond to male hormone irrespective of whether this is provided by stimulating the testicle to produce it through gonadotropic substances, or by administering it subcutaneously through injection. In either case the prostates become greatly enlarged but it is a functional enlargement, not the typical non-functional hypertrophy of the senile man. I am not offering any suggestion relative to treatment of prostatic hypertrophy by the male hormone.

In regard to testicular descent I believe that the testis hormone is a decided factor but rather than administering it as such it appears at present preferable to employ gonadotropic substances to induce the intact testis to secrete the hormone under stimulation. I am not acquainted with an active male hormone preparation on the market. The yield from testis tissue or urine is small and to maintain a castrated male rat with normal accessory reproductive organs requires the daily injection of the total effective extractives from approximately fifty grams of tissue each day. In the rat it is difficult to perform the operation for experimental retention of the testis in the abdomen so effectively that it will remain undescended while large quantities of male hormone are present. The scrotum becomes enlarged and the canals tend to enlarge and reopen. In testicular undescent it would appear advisable to try injections of gonadotropic substances before operation for scrotal placement. Dr. Sexton of St. Louis has reported some success from such treatments. Obviously the condition of the organ itself, whether adherent, or with shortened cord, introduces certain limitations to the treatment.

Replying to Dr. Albright's question regarding the best method for detecting the amounts of hormone present we have used in the rat both the prostate and seminal vesicle tests. The prostate gland reacts to smaller amounts of substance than do the seminal vesicles and we have found in some cases an apparent normal prostate but seminal vesicles of a castrate type. Normality of the seminal vesicles requires the presence of greater amounts of hormone and where these are normal the prostate will always be found to be normal. A combination of the two tests in each animal has much to recommend it.

The injurious effects of gonad hormones on the gonads were illustrated particularly by reference to the effects of estrin in the normal male. Such treatment caused severe injury to the testicle, but so does the injection of testis hormone. Estrin injections into normal females injure the ovaries but

stimulate the uterus. The only explanation for such injuries I have been able to offer is the one common to the two conditions, namely an injury to the hypophysis by excessive amounts of the hormone of either sex and the consequent gonad injury from defective amounts of hypophysial secretion.

Dr. Merritt raises the question of permanent damage to ovaries of normal females from estrin injections. In all cases where the problem has been followed subsequent to cessation of estrin injection, the animals (rats) have returned to a reproductive state; the damage unless decidedly severe is probably not permanent.

Dr. Colby asks about the relation of male hormone to sterility. So far as we know, male hormone is entirely ineffective in cases of sterility. What apparently is defective is pituitary activity and we do not yet know how to stimulate the pituitary. Work by Rowan in Alberta has shown that gradual daily increases in the length of the day (by additions of electric light at dusk) have been effective in causing the testes of birds to resume activity and to produce spermatozoa in mid-winter. Shortening of the day period of light led to reduction of this high peak of activity. Environmental influences (light periods or other factors) may be found effective measures for hypophysial stimulation but our knowledge of these matters is at present very limited. I do not know how to remedy those cases of borderline sterility by hormone treatment, but rational experimentation, a critical attitude, and continued efforts rather than sporadic ones are to be recommended as a means of advancing our knowledge of these conditions.

CONDENSED DIRECTIONS TO PATIENTS ON HOW TO LIVE WITH ANGINA PECTORIS

It is often possible to live a moderately active life of normal duration in spite of your trouble, provided you adopt certain simple rules of living.

1. Cultivate an optimistic nature as to the future. Do not place reliance on statements, and do not accept advice from others than your doctor.

2. The measure of how much exercise and work you may do is determined by how much is required to produce your distress. Do just a little less than produces your pain. Excessive physical strain even for a short time may produce serious complications.

3. Avoid emotional upsets like anger, worry, fretting, and even exciting social life. Even joyful excitement, if excessive, may be harmful. Do not let minor things irritate and disturb you.

4. Your diet will be regulated by your physician. If you are obese, reduction diet will accomplish a great deal for you. If you are of normal weight, avoid heavy, hearty meals. Better eat five light meals than three heavy ones. No particular food need be omitted unless the doctor tells you so. Rest for fifteen to thirty minutes after each meal. Avoid banqueting. One overeating bout may cause serious trouble.

5. Tea and coffee are seldom of importance unless taken to excess. Whiskey, or brandy, in moderate doses is often helpful, seldom harmful. Do not drink large quantities of fluids at one time. Tobacco in large amounts is harmful to most everyone, in moderate amounts to many, and even in small amounts to some. It is well to be very moderate in its use, or abstain from it entirely.

6. Rest periods though of only short duration are of great help. Frequent vacations even for a few days are very valuable. A Saturday afternoon or a Sunday in bed, retiring early at night, lying on a couch after meals, or just lounging about the house, are different methods of resting. Your circumstances will suggest special times and methods of resting.

7. Avoid constipation with straining at stool. If you have difficulty in urination, tell your doctor about it. Sexual intercourse is usually better omitted.

8. Do not rush after trains or to keep appointments. Plan so that such haste will be unnecessary. Do not walk against high winds, up steep hills or up long stairs. Do not shovel snow, dig in gardens, or do heavy work. Avoid exposure to extreme cold, and travel if possible in closed vehicles during zero weather. Do not indulge in competitions in whatever form of play you attempt. Avoid exercise or work in the heat of the day and with exposure to the sun. Sun baths have no beneficial influence on your trouble and the excessive heat is exhausting.

9. Enjoy the pleasures of social life, the table, the outdoors, and your work, within your limitations.

10. Always carry your nitroglycerine tablets with you. If you follow the directions in the preceding paragraphs, you will seldom need them, but do not hesitate to take them freely as ordered by your doctor.

11. If you are planning dental or other surgery, talk it over with your doctor, or if you are planning long journeys or unusual tasks, get his advice before undertaking either.

12. If at any time your pain is unusually severe, and is not relieved by the usual procedures, go to your home in the manner involving the least muscular exertion, preferably by taxicab, go immediately to bed and send for your doctor. Do not accept indigestion as an explanation for the persistent pain.

JOHN SPROULL, M.D.

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Haverhill, Massachusetts.

NOTE: Copies in booklet form may be obtained at the cost price of three cents per copy plus postage on application to Dr. Sproull.

THE VISUAL MECHANISM IN DIABETES MELLITUS

(A Comparative Study of 2002 Diabetics, and 457 Non-Diabetics for Control)

BY J. HERBERT WAITE, M.D.,* AND WILLIAM P. BEETHAM, M.D.*

(Continued from page 379, Issue of February 28, Volume 212, Number 9)

It is difficult to decipher the rôle played by iridescent crystals in the lens. In diabetics, we found bodies resembling cholesterin crystals in the anterior cortex of 468 lenses (11 per cent), and in the posterior cortex of 316 lenses (8 per cent). Of seventy-five diabetics with blood cholesterol above 229 mg. per 100 cc., only seven patients (9 per cent) showed visible iridescent crystals in the anterior cortex of the lens. In the non-diabetics, we found similar bodies in the anterior cortex of ninety-seven lenses (10 per cent), and in the posterior cortex of thirty-nine lenses (4 per cent), Plate IX. In 110 children with diabetes, White and Hunt[29] found that abnormally high cholesterol in the blood was an exception in uncomplicated diabetes, that there was no close correspondence between hyperglycemia and hypercholesterinemia, and that increase of cholesterin in the blood of children is regularly observed only in overnutrition and in coma. In the 297 juvenile diabetics of this series, all under the age of twenty years, we found iridescent crystals in the lenses of twenty-nine, and in six of these patients cataract was associated.

c. *Complicated cataract.* The opinion has often been expressed in the literature that cataracta complicata are more frequent in diabetics than in non-diabetics. Our figures would indicate that cataracta complicata are almost equally distributed between the two groups.

CATARACTA COMPLICATA—IN DIABETICS AND NON-DIABETICS

(DISTRIBUTION BY AGE)

Diabetics Eyes Exam.	Diabetics Cataracta Complicata	Diabetics %	Age	Non-Diabetics %	Non-Diabetics Cataracta Complicata	Non-Diabetics Eyes Exam.
130	0		Under 10			
464	11	2%	10-19			
284	12	4%	20-29		0	20
319	8	2%	30-39	1%	2	130
520	16	3%	40-49	3%	9	306
1032	55	5%	50-59	10%	24	232
999	102	10%	60-69	18%	31	170
253	42	16%	70 up	16%	9	56
4001	246	6%		8%	75	914

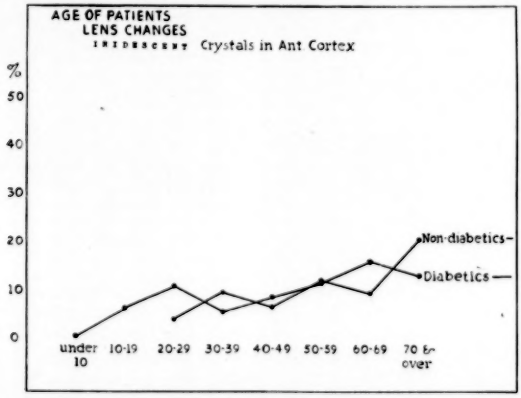

PLATE IX.

In diabetics eighty per cent, in non-diabetics eighty-five per cent of all complicated cataracts were found in patients over fifty years. The average age of the diabetics with complicated cataract was fifty-eight years, while that of the non-diabetics was sixty years.

We are inclined to think of complicated cataract as associated with disease of the posterior

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segment of the globe, or with toxic ocular fluids. Diabetics with complicated cataract exhibit no obvious correlation between the lens changes and the duration of diabetes or the amount of insulin administered, or between the blood sugar content or the presence of so-called diabetic retinitis. An average blood sugar content of 234 mg. per 100 cc. was found for 102 diabetics having complicated cataract*, and of these there were sixteen patients (15 per cent) with a maximum fasting blood sugar above 300 mg. per 100 cc. Among 103 recorded NPN in diabetics with complicated cataracts, only seven patients had values over 44 mg. per 100 cc. (7%). Among the 123 diabetics with complicated cataract, the fundi were visible in 111 patients, among whom deep retinal hemorrhages were found in forty-four (39 per cent), and superficial retinal hemorrhages in thirteen (10 per cent). In this same group of 123 diabetics with complicated cataracts, we find eleven cases of gangrene (9 per cent), eighteen cases of neuritis (14 per cent), five cases of coma (4 per cent), and two cases of syphilis (1 per cent).

CATARACTA COMPLICATA—IN DIABETICS

(Duration of Diabetes)

Duration Diabetes	Av. Age Each Duration Group	Eyes Exam.	Cataracta Complicata	%
Less 1 yr.	40.1	1001	26	2%
1-1.9 yr.	45.1	452	27	6%
2-2.9 yr.	45.1	378	33	8%
3-3.9 yr.	45.1	560	54	9%
5-9.9 yr.	41.2	966	52	5%
10-15	55.2	419	31	7%
15 up	61.8	225	23	10%
		4001	246	

CATARACTA COMPLICATA IN DIABETICS—AMOUNT OF INSULIN

Amt. Units Insulin	Av. Age Each Group	Eyes Exam.	Cataracta Complicata	%
None	56	697	35	5%
1-14	54	1004	75	7%
15-29	47	1366	92	6%
30-50	37	750	29	4%
50 up	26	184	15	8%
		4001	246	

Of course, complicated cataract does go on to maturity, as evidenced by the finding of fissures, vacuoles, and spokes in fifty per cent of all complicated cataracts recorded. In the mature cataract it is impossible to define the primary type by slit-lamp or any other means, and hence many complicated cataracts may escape proper enumeration.

*Of 123 diabetics with complicated cataracts, blood sugar determinations were available in only 102 patients.

d. *Flocculi.* On account of the resemblance to flocculent chemical precipitates of anterior and posterior cortical lens opacities in juvenile diabetics, we have learned to speak of opacities of this type as "flocculi". Similar opacities have been described as "diabetic cataract" by Schnyder[3], and by Goulden[4], and as "snowflake" opacities by O'Brien, Molsberry and Allen[5].

Among 297 juvenile diabetics of this series, we found with the aid of the slit-lamp eleven patients with flocculi, or about four per cent. Flocculi are always bilateral, and situated within the anterior cortical levels, but never in the nucleus between the Y sutures. In seven of our eleven cases, fine iridescent crystals were associated with the flocculi, and in the same levels of the lens. While flocculi may first appear just beneath the capsule of the lens, they tend to increase by invading the cortex more deeply, and they are frequently associated in the course of slowly progressive development, as in eight of our eleven cases, with posterior cortical degeneration, warranting inclusion under the term complicated cataract. One of our eleven cases of flocculi succumbed in coma. In six of the ten survivors, surgery has been applied to remove the cataractous lens or lenses.

The incidence of flocculi found in any series may depend upon many factors, such as the diligence employed in the slit-lamp search, the number of patients induced to attend on account of visual disturbances, and others. The earliest appearance of flocculi may be entirely missed in ophthalmoscopic examinations, and may exert no adverse effects upon visual acuity. Among twenty instances of cataract found in 126 patients studied, O'Brien, Molsberry and Allen found twelve "snowflake" cataracts, or about ten per cent incidence, but they also note that severe, prolonged, and poorly controlled diabetes was the rule in the cases with such lens changes. In half of our patients presenting flocculi, diabetes was poorly controlled at the time the patient first came under supervision. In the remainder of our patients presenting flocculi, the characteristic opacities appeared and multiplied in spite of the most satisfactory control of diabetes. One of these latter cases is well represented in case history No. 9535.

Case No. 9535

In October, 1930, the patient, S. B., a girl aged twelve years, was first examined. At this time, the duration of diabetes was seven months, with insulin therapy for a few days only. There was no history of coma. X-rays disclosed a heart of normal size, and no sclerosis of the radial vessels. X-ray of the chest for tuberculosis showed suspicious shadows. The blood pressure was 100/64. The blood Wassermann test was negative. The maximum blood sugar, fasting, was 270 mg. per 100 cc.; the maximum, non-fasting, was 300 mg. The minimum blood sugar was 80 mg. NPN examined twice was 35 and

37 mg. There were no casts or albumin in the urine, and the urinary sugar decreased from 8.0 per cent to 0.2 per cent during the patient's hospital stay. On discharge from the hospital, the insulin dosage was 34 units daily. No basal metabolism test was done.

The eye findings were as follows: the refraction, with homatropine cycloplegia, was:

Rt. eye: +3.25 sphere and +.75 cylinder at axis 90° =6/5
Lt. eye: +3.25 sphere and +.50 cylinder at axis 90° =6/5

The accommodation measured 14. diopters in each eye. Maddox rod, prism duction, stereopsis, associated movements, convergence power, and accommodation were normal. Examinations by slit-lamp and ophthalmoscope were negative. The peripheral visual fields were full, and the blind spots of normal size. Intraocular pressure registered 31 mm. in each eye by Schiötz.

In September, 1931, the patient was again examined, and gave a history of many severe colds. At this time, the blood pressure was 104/78. The maximum blood sugar, non-fasting, was 140 mg. per 100 cc. There was no trace of sugar in the urine. When discharged from the office, the patient was advised to use 29 units of insulin daily.

The eye findings at this time were as follows: refraction, with homatropine cycloplegia, was:

Rt. eye: +2.25 sphere and +.50 cylinder at axis 90° =6/5
Lt. eye: +1.75 sphere and +.25 cylinder at axis 90° =6/5

The accommodation measured 14. diopters for each eye. Examination by slit-lamp disclosed 1 iridescent crystal in the posterior cortex of the right eye, and 2 vacuoles in the posterior cortex of the left eye. The fundus examination was negative. Visual fields were full, and the blind spots were of normal size. The intraocular pressure registered 28 mm. in each eye by Schiötz.

In December, 1931, the patient gave a history of poor vision in the left eye over a period of two weeks; the urine had not been sugar free during that time. The heart was of normal size to percussion. An x-ray of the chest again gave shadows suspicious of tuberculosis. The blood pressure was 100/80. Maximum blood sugar fasting was 130 mg. per 100 cc.; maximum blood sugar non-fasting was 220 mg. The minimum blood sugar was 80 mg. The NPN was 32 mg. Blood cholesterol was 189 mg.; blood calcium 11.0 mg., and phosphorus 4.3 mg. Phenolsulphonephthalein excretion was 40 per cent. The urine albumin was negative, and the urinary sugar fell from 1.8 per cent to 0.1 per cent during hospitalization. The patient was taking 29 units of insulin daily on discharge.

The eye examination at this time disclosed the following facts: refraction, by homatropine cycloplegia, was:

Rt. eye: +1.50 sphere and +.50 cylinder at axis 90° =6/5
Lt. eye: —. 75 sphere =6/6

The accommodation was 12 diopters in each eye. Examination by slit-lamp showed that the anterior cortex of each lens was now filled with very fine flocculi, more marked in the left, with many associated iridescent crystals. The secondary suture lines in the anterior cortices were visible, and several large vacuoles were visible in the posterior cortex in polar position. The fundi were normal. Intraocular pressure registered 31 mm. in each eye by Schiötz.

In March, 1932, the patient was again examined, at which time the vision in the left eye was still reported as poor. Blood cholesterol was 171 mg., and the daily insulin dosage was 34 units.

The homatropine refraction was:

Rt. eye: + .75 sphere and +.50 cylinder at axis 90° =6/5
Lt. eye: —1.75 sphere and +.50 cylinder at axis 90° =6/7

No changes were noted in the slit-lamp picture. The intraocular pressure was 22 mm. in each eye, Schiötz, and the fundi normal.

In September, 1932, when the patient reported, she gave a history of having received dionin in May from Dr. Allen Greenwood, and of marked improvement in vision of the left eye during the subsequent few weeks.

The refraction at this time was:

Rt. eye: +1.00 sphere and +.50 cylinder at axis 90° =6/6
Lt. eye: +1.50 sphere =6/6

No change was indicated in the slit-lamp picture. Intraocular pressure was 22 mm. in each eye, Schiötz.

In December, 1932, the patient's vision was reported as better. The heart was normal to percussion, but slightly large to x-ray. No sclerosis of the radial vessels was noted. Slight calcification was shown opposite the left ankle joint by x-ray, and the x-ray of the chest still revealed shadows suspicious of tuberculosis. The blood pressure was 90/60; the blood cholesterol 203 mg., and the urinary sugar was found to be 0.2 per cent. The insulin dosage at this time was 52 units daily.

A study of the eyes by slit-lamp revealed no changes from those noted at previous visits. The intraocular pressure was 25 mm. in each eye, and the fundi normal.

In April, 1933, with daily insulin therapy of 59 units, the maximum blood sugar, non-fasting, was 360 mg. per 100 cc. Blood cholesterol was 206 mg.; urinary sugar 5.2 per cent.

The refraction was:

Rt. eye: +1.50 sphere = 6/6
Lt. eye: +1.75 sphere = 6/7

There was no change in the slit-lamp picture, and the intraocular pressure was 22 mm. in each eye.

In July, 1933, the heart size of the patient was found to be normal to percussion. The blood pressure was 110/70. The maximum blood sugar was 110 mg., NPN 28 mg., and blood cholesterol 182 mg. per 100 cc. The urine of the patient was sugar free. Insulin dosage was 55 units daily.

The patient's visual acuity was 6/6 minus in both eyes. The slit-lamp findings were unchanged. The retinal vessels were normal, and the intraocular pressure measured 22 mm. right and left.

Whether the incidence is 4 per cent or three times that figure, the presence of bilateral slowly progressive lens opacities of the type noted in juvenile diabetics is a distinctly abnormal finding. Whether the opacities are the direct outgrowth of diabetic processes is another question, and one that cannot be solved without considerably more data than we possess at present. In our eleven patients with flocculi, there present a striking collection of abnormalities of body and of eye. All of them are severe diabetics with

heavy insulin requirements, and with wide and precipitate shifts in blood sugar. Three of them have had coma, one fatally. Six of the eleven present evidence of endocrine disturbance apart from diabetes. Three of the eleven had a blood NPN above 39 mg. per 100 cc., and one had a blood cholesterol over 229 mg. per 100 cc. Nine of the eleven had demonstrable sclerosis of peripheral or of retinal vessels, and one had a blood pressure of 160 systolic, and 100 diastolic. One must not forget that opacities of the flocculi type have been found in endocrine disorders, notably in parathyroid tetany, scleroderma, and myotonic dystrophy. It would seem desirable, therefore, to withhold judgment about the real etiology of flocculi cataracts until sufficient data are forthcoming about lens physiology-pathology.

NUCLEAR CHANGES

	4001 Eyes of Diabetics	%	914 Eyes of Non-Diabetics	%
Embryonic and Fetal Nucleus				
Clusters between Y's	464	11%	250	27%
Dust between Y's	103	2%	46	5%
Y suture opacities	7		0	
Zonular Cataract	2		0	
Adult Nucleus				
Dots outside Y's	157	4%	60	6%
Mild sclerosis	761	19%	211	23%
Sclerosis	109	2%	6	
Protuberances	46	1%	6	

FLOCCULI

(Bilateral Cortical Opacities in Juvenile Diabetics)

Age	Yrs. Dur. DM.	Amt. Ins.	Coma	Infections	Per. Ves. Scl.	Ret. Ves. Scl.	Bld. Sug. Shft.	Max. NPN	Max. Chol.	Calc.	Phos.	Endocrine	Retinitis	Comp. Cat.	Operations
S. B. 12	1.7	29 59	0	TB?	X	0	360 60	36	206	11.0	4.3	0	0	no	no
O. C. 18	1.0	36 51	0	0	0	R	520 40	41	362	10.8	3.4	T —16	0	yes	yes
C. F. 13	2.3	26 54	0	0	0	R	320 80	32	214	10.3	3.6	0	0	yes	yes
M. M. 23	7.0	62 65	4x	TB Carb.	X	R	330 80	39	202	9.8	3.8	T?	R	yes	yes
J. L. 27	5.5	44	0	0	R	0	250 40	42	192	9.8	3.4	P?	0	yes	yes
W. G. 18	5.0	34 52	1x	TB Carb.	0	0	710 80	37	208	9.8	3.7	0	0	yes	yes
E. S. 27	7.5	24 34	0	0	R	0	360 30	35	132			0	0	yes	yes
J. S. 16	8.5	24	0	0	0	R	380 60	38		11.2	4.3	P	R	no	no
E. T. 16	3.0	42	0	0	R	R	180 80	29	200	10.4	3.8	0	0	yes	no
E. W. 13	2.5	27 54	0	0	R	0	270 50	33	185	11.1	2.0	P	0	yes	no
R. Y. 15	6.0	53	1x Death	0	0	0	320 70	32				T +26	0	no	Death

VIII.—B.—3. *Nuclear Changes*

Nuclear lens changes will be considered in three categories, following the lens architecture revealed by the slit-lamp method: opacities of the embryonic and fetal nucleus, zonular cataract, and opacities of the adult nucleus.

Nuclear lens opacities in either diabetic or non-diabetic groups would seem to throw no light upon the rôle diabetes may play in the production of cataract, because opacities of this type are usually laid down before birth. Zonular cataract was found only twice, and it merits no discussion here. Opacities of the adult nucleus are related to the uniformity and degree of sclerosis, in which there seems to be no outstanding difference in the two groups.

VIII.—B.—4. *Miscellaneous Lens Changes*

Under miscellaneous changes in the lens will be considered:

a. Capsular opacities.
b. Coronary opacities.
c. Aphakia

MISCELLANEOUS LENS CHANGES

	4001 Eyes of Diabetics	%	914 Eyes of Non-Diabetics	%
Anterior Capsule				
Congenital pigment deposits	50			
Anterior capsular cataract	10			
Exfoliation zonular lamella	3			
Posterior Capsule				
Hyaloid remnants	39			
Coronary Opacities	512	12%	168	18%
Aphakia				
(40 cataracts extracted subsequent to examination, therefore, not counted here)	22		7	

We wish to enter miscellaneous lens opacities for record only, and we do not wish to imply that there is any relationship between such opacities and diabetes.

IX. The Vitreous

The following vitreous abnormalities were found:

VITREOUS ABNORMALITIES

	3407 Eyes of Diabetics Over 20 Years Old		914 Eyes of Non-Diabetics Over 20 Years Old	
Vitreous opacities { few 185 / many 399	584	17%	128	14%
Retinitis proliferans, Pre-retinal hemorrhage (31 patients)	49	1.4%	1	
Vitreous hemorrhage	8		0	
Asteroid hyalitis	5		2	

A most interesting group is to be found in thirty-one diabetics showing forty-nine eyes with pre-retinal hemorrhages or with proliferation of vessels and connective tissue in retina and vitreous. The age distribution of this group was as follows:

40-49 years— 5 patients
50-59 years—16 patients
60-69 years—10 patients

31 total

In eighty-three per cent of this group, the retinal arteries were graded No. 3 or over, and the retinal veins showed conspicuous changes suggesting an etiological rôle. In ninety per cent of this group there was one or more of the following findings:

Systolic pressure over 160 mm.—19 patients
Diastolic pressure over 90 mm.—15 patients
Enlarged heart— 20 patients
Angina-coronary disease— 5 patients
NPN above 40 mg. per 100 cc.—19 patients
Albuminuria— 20 patients
Phthalein excretion under 30%— 9 patients
Gangrene— 5 patients

Thus it would seem that this group is derived from diabetic patients with cardio-vascular-renal complications, and with outspoken disease of the retinal vascular tree.

The part played by diabetes alone in this group seems to be trivial. Only twenty-nine per cent of this group showed fasting blood sugar over 250 mg. per 100 cc. Ninety-three per cent of the insulin-treated group received an average daily dose of insulin of 17 units, and twenty-nine per cent of the entire group received no insulin whatever.

This group is characterized also by the frequency of pupillary abnormalities. Of the forty-nine eyes listed, fourteen eyes showed pupils which presented sluggish response to light, failed to respond to light, or failed to dilate normally with mydriatics. In none of the fourteen eyes could any obvious reason be found by slit-lamp for abnormal pupillary behavior. All fourteen eyes showed good light perception, and half of them attained a corrected visual acuity of 6/12 or better.

In our experience the prognosis for sight in this group is uniformly poor, because, in spite of the best diabetic handling, hemorrhages tend to recur spontaneously and to organize. In one of these patients, we were able to observe the development of a progressive retinal detachment brought about by contraction of proliferated tissue in the vitreous. Eyes of this type are exceedingly poor surgical risks. We cannot agree with Grafe[11] that insulin predisposes to hemorrhage in eyes of this type, and we suspect that large doses of potassium iodide may be distinctly harmful in eyes with such badly diseased vessel walls.

X. The Retina

The following chief retinal abnormalities were found:

RETINAL ABNORMALITIES

	Diabetics in 3915 Visible Fundi		Non-Diabetics in 101 Visible Fundi	
Deep Retinal Hemorrhages	730	18.6%	34	3%
Waxy Exudates	420	10.7%	7	.7%
Nerve Fiber Layer Hemorrhages	196	5.0%	33	3%
Cotton Wool Exudates	168	4.3%	35	3%
Iridescent Crystals	28	0.7%	4	
Proliferation of Capillaries in Retina	26	0.7%		

Since the time of Jaeger, there has been no dispute about the frequency of deep retinal hemorrhages and associated waxy exudates in the retinae of diabetics past middle age, but there has been much discussion about causes of these retinal changes. Proof of causation of deep retinal hemorrhages requires more than observed association with hypertension or arteriosclerosis. We may no longer think of parallelism between the hydrodynamics within the larger vessels and the hydrodynamics within the capillaries, because the capillaries are subject to separate and purposive control. The observed abnormalities in the walls of the larger retinal vessels should no longer be cited to explain the origin of the deep retinal hemorrhages, because all evidence points to the capillaries as the source of these hemorrhages. To explain the source of deep retinal hemorrhages, we should seek more information about the physiology and pathology of the retinal capillaries, about their normal and abnormal hydrodynamics, about glycogen deposits which are known to affect other ocular tissues, about the normal and abnormal permeability of capillary walls, about thromboses which may form within them, and about toxins which may affect them adversely. Until this knowledge is forthcoming, either from clinical studies or from the study of laboratory animals, we can form no valid opinion about the source of deep retinal hemorrhages in diabetics.

Consequently, we have made no attempt to classify types of retinitis in diabetics, types which merge into one another, and types which permit of no clear-cut interpretation in terms of the visible retinal vascular tree.

For our purposes, we have been content to seek diligently and to record the retinal abnormalities to be found in diabetics and non-diabetics, so that correlation might be attempted with various factors commonly incriminated. To this end examination of each fundus was made with dilated pupil by two observers working independently. In two-thirds of our cases, the hemorrhages were sufficiently numerous so that it is difficult to conceive of overlooking them. In one-third of our cases, the hemorrhages were so few that a careful and tireless search was required to find them. Herein probably lies the explanation of the incidence of deep retinal hemorrhages of eighteen per cent in 2002 diabetics here reported, as contrasted with hemorrhage incidence in other large diabetic series (Spalding & Curtis 5 per cent, McKee 6 per cent, Mayo Clinic 9 per cent, Gray 11 per cent).

It would seem desirable to present our analyses of deep retinal hemorrhages according to the following scheme:

a. Deep retinal hemorrhages in relation to age.
b. Deep retinal hemorrhages in relation to sclerosis of retinal vessels.
c. Deep retinal hemorrhages in relation to hypertension.
d. Deep retinal hemorrhages in relation to renal disease.
e. Deep retinal hemorrhages in relation to duration of diabetes.
f. Deep retinal hemorrhages in relation to insulin.
g. Deep retinal hemorrhages in relation to blood calcium.
h. Deep retinal hemorrhages in relation to blood sugar.

X.—1.—a. *Incidence of Deep Retinal Hemorrhage By Age*

DEEP RETINAL HEMORRHAGES—IN RELATION TO AGE

Age Group	Diabetics			Non-Diabetics		
	Visible Fundi	Both Eyes Deep Hems.	% of Fundi Visible	% of Fundi Visible	Both Eyes Deep Hems.	Visible Fundi
Under 10	130	0				
10-19	464	4	.8%			
20-29	279	10	3.2%		0	20
30-39	315	17	5.4%	4.6%	6	129
40-49	514	75	14.5%	2.6%	8	305
50-59	1006	282	28.0%	6.1%	14	229
60-69	966	270	28.0%	3.6%	6	164
70 up	241	72	30.0%		0	54
	3915	730	18.0%	3.0%	34	901

This table shows strikingly that deep retinal hemorrhages are rare in the younger diabetics, however severe the diabetes may be, and that they become more and more frequent in older diabetics, Plate X. The average age for all diabetics with deep retinal hemorrhages was fifty-eight years. Of deep retinal hemorrhages found in 730 diabetics, only four per cent were found in patients under forty years old, ten per cent were found in patients from forty to fifty years old, and eighty-six per cent were found in patients over fifty years old.

X.—1.—b. *Deep Retinal Hemorrhages In Relation to Sclerosis of Retinal Arteries*

Two types of sclerosis have been found histologically in retinal arteries. In the larger vessels on the disc, the atheromatous type of change involving the endothelium is the rule, and it may lead to occlusion of the affected vessel. In the smaller retinal branches, which contain no elastic lamina, the more common type of sclerosis is the hyalin thickening of the media (Mönckeberg), without complete obliteration of the lumen. It is very difficult to differentiate the two histological types by clinical methods. At the arteriovenous crossings, both artery and vein may be involved in the common sclerotic process, as Friedenwald[30] has pointed out.

For clinical purposes, it has seemed desirable to grade all retinal arteries according to the following scheme:

Grade 0—no abnormalities

Grade 1—increased light reflexes

Grade 2—prominent light reflexes
slight loss of transparency of vessel walls
slight arteriovenous compression

Grade 3—moderate loss transparency of vessel walls
moderate arteriovenous compression
irregularity in caliber of vessels

Grade 4—arterial walls opaque
sheathing of vessels
obstruction of lumen

The grade given to 3893 retinal arterial trees of diabetics is shown in the following tabulation, with an average sclerosis indicated for each decade of age.

We were unable to demonstrate conclusively

RETINAL VS. RADIAL ARTERIOSCLEROSIS IN DIABETICS

Clinical Grade of Sclerosis	Radial Vessels* Rt. Only	%	Retinal Vessels† Rt. & Lt.	%
No. 0	455	24%	1042	26%
No. 1	498	27%	905	23%
No. 2	677	37%	1294	33%
No. 3	166	9%	595	15%
No. 4	33	1.8%	57	1.4%
	1829		3893	
Unrecorded	173		108	
	2002		4001	

3 anophthalmos

*Rating by Joslin group.
†Rating by Waite and Beetham.

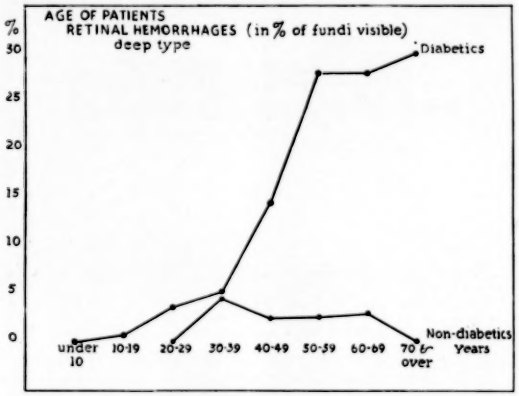

PLATE X.

SCLEROSIS OF RETINAL ARTERIES IN DIABETICS, BY AGE DECADES

Grade of Sclerosis	130 Under 10	464 10-19	274 20-29	315 30-39	512 40-49	1001 50-59	957 60-69	240 Over 70	= 3893
No. 0	130	451	253	153	49	6			= 1042
	100%	97%	92%	48%	9%	.6%			= 26%
No. 1		11	13	130	260	322	150	19	= 905
		2%	4%	41%	51%	32%	16%	8%	= 23%
No. 2		2	8	30	177	458	508	111	= 1294
		.4%	3%	10%	34%	46%	53%	46%	= 33%
No. 3				2	20	196	276	101	= 595
				.6%	4%	19%	29%	42%	= 15%
No. 4					6	19	23	9	= 57
					1%	2%	2%	4%	= 1.4%

any preponderant sclerosis of retinal arteries in diabetics, over those of non-diabetics of this series. We consider that isolated instances fail to prove the contention.

While the grade of sclerosis given by us to retinal arteries of diabetics shows striking parallelism to that given by the Joslin group for radial arteries, as shown in the table above, and in Plate XI, there were patients in whom no such correlation existed. In these patients, sclerosis is capricious and manifests itself markedly in some vessels, and spares others.

The number of deep retinal hemorrhages tabulated against the degree of sclerosis found in 1944 right eyes of diabetics are shown in the following table:

DEEP RETINAL HEMORRHAGES AND SCLEROSIS RETINAL ARTERIES IN DIABETICS

Grade of Sclerosis of Retinal Vessels	Right Eyes Diabetics	Right Eyes Hemorrhages	%
No. 0	519	1	
No. 1	460	49	10%
No. 2	638	165	25%
No. 3	299	133	44%
No. 4	28	22	79%
	1944	370	

X.—1.—c. *Deep Retinal Hemorrhages in Relation to Arterial Hypertension*

Deep retinal hemorrhages were found in 730 eyes of the 372 diabetics of this series. Systolic arterial pressures were recorded in 369 of these patients, and diastolic pressures in 366. In the patients with pressures recorded, Plates XII and XIII show that deep retinal hemorrhages were present in fifty-four per cent of the diabetics that had a systolic pressure under 160 mm. mercury, and in sixty-two per cent of the diabetics that had a diastolic under 90 mm.

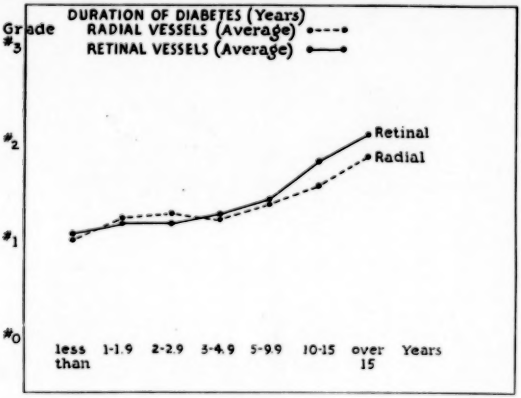

PLATE XI.

While hemorrhages seem to increase with high grades of sclerosis in the larger retinal vessels, it does not follow that such vascular sclerosis causes the hemorrhages. One must consider in this connection the unescapable increase in age of patients, with tissue senility and altered capillaries.

The relationship between retinal vessel sclerosis in diabetics, and abnormally high blood constituents is shown in the following table:

BLOOD SUGAR, NPN, AND CHOLESTEROL AGAINST SCLEROSIS OF RETINAL VESSELS IN DIABETICS

Grade Sclerosis Retinal Vessels	Blood NPN Over 44 mg.	Blood Cholest. Over 230 mg.	Blood Sugar Over 300 Fasting Over 400 Non-Fasting
No. 0	12	35	118
No. 1	11	13	57
No. 2	29	19	63
No. 3	18	4	27
No. 4	2	0	1
	72	71	266

Diabetics with vessels recorded, and blood constituents recorded.

mercury. Thus, considerably more than half of the hemorrhages occurred in patients with blood pressures falling within the normal and high normal range. Furthermore, if plotted by duration of diabetes according to Plate XIV, the curve for deep retinal hemorrhages does not follow the same trend as do the curves for systolic pressure over 160 mm. mercury, or diastolic pressure over 90 mm. mercury.

X.—1.—d. *Deep Retinal Hemorrhages in Relation to Renal Disease*

Tangible evidence of renal disease is nitrogen retention reflected in blood nonprotein nitrogen

DEEP RETINAL HEMORRHAGES AND BLOOD NPN IN DIABETICS

NPN	Diabetics	Diabetics with Deep Ret. Hems.	%
Less than 35 mg.	877	161	18%
35-39 mg.	450	85	18%
40-44 mg.	180	50	27%
45-50 mg.	56	14	25%
Above 50 mg.	20	6	30%
	1583	316	

analyses. Nonprotein nitrogen determinations were made from the blood in 316 of the 372 diabetics with deep retinal hemorrhages, and in 1583 of the 2002 total diabetics.

Of the 316 diabetics with deep retinal hemorrhages and NPN determinations, only twenty (6 per cent) showed values over 44 mg. blood NPN per hundred cubic centimeters of blood. If blood NPN be plotted according to duration of diabetes, and deep retinal hemorrhages be similarly plotted, there is no obvious analogy. Plate XV.

X.—1.—e. *Deep Retinal Hemorrhages in Relation to Duration of Diabetes*

Plate I and Plate II reflect our reasons for believing that diabetes is more severe during the first three decades of life; the blood sugar levels are consistently higher, and the insulin requirements are consistently greater than at any

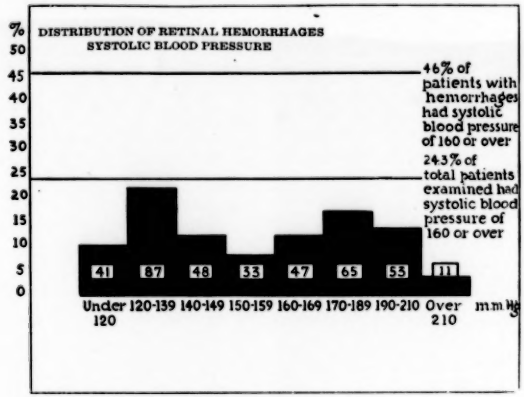

PLATE XII.

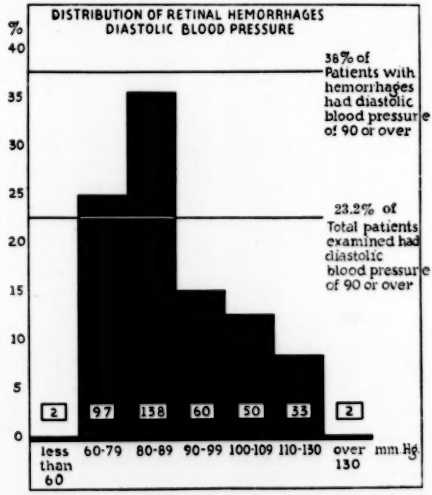

PLATE XIII.

other time in life. The deep retinal hemorrhage situation is just the reverse. During the first three decades of life of the diabetics in this series, deep retinal hemorrhages were found in only two per cent of the total eyes or in fourteen eyes of 730 with hemorrhages. As the diabetes grows milder with each increasing decade, the hemorrhages become more numerous.

There is with increasing duration of diabetes an increasing incidence of deep retinal hemorrhage, out of proportion to the accompanying increase in the age of the patient as reflected in the average for each group. Plate XVI.

Insulin has repeatedly been named as a potential cause for retinal hemorrhage in diabetics. If we sort the diabetic material on the

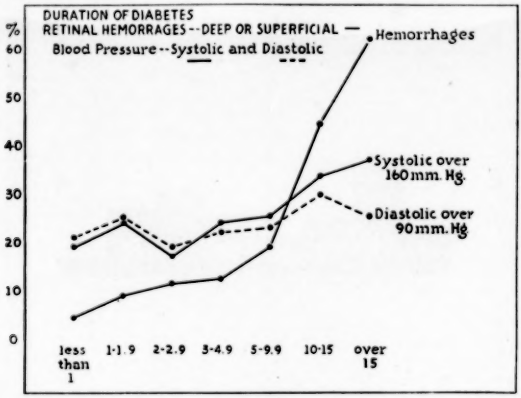

PLATE XIV.

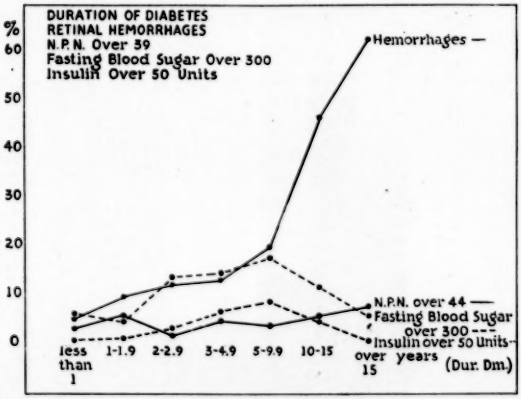

PLATE XV.

RETINAE OF DIABETICS—DEEP RETINAL HEMORRHAGES—DURATION DIABETES

3915 Fundi Visible	993	448	370	549	935	403	217
Duration Diabetes	1 yr.	1-1.9	2-2.9	3-4.9	5-9.9	10-15	Over 15
Deep Hemorrhages	57	48	44	76	192	185	128=730
Average Age	40	45	45	45	41	55	62
	5.7%	10.7%	11.8%	13.8%	20.5%	43.1%	58.9%

X.—1.—*f. Deep Retinal Hemorrhages in Relation to Insulin Fundi Visible*

DEEP RETINAL HEMORRHAGES IN RELATION TO INSULIN FUNDI VISIBLE

Av. Age	% Fundi Visible	Total Diab. Eyes	Insulin	Deep Retinal Hemorrhages	% Hems.
56	19%	678	None	132	18%
54	24%	978	1-14	238	32%
47	18%	1339	15-29	242	33%
37	14%	739	30-50	105	14%
26	7%	181	50 up	13	2%
		3915		730	

diabetics received blood calcium (total) determinations, and the results were as follows:

Age of Group	Total Blood Calcium
10-19	11.4 mg. per cent
10-19	9.4
30-39	8.2
40-49	10.7
50-59	10.4
60-69	11.0
60-69	10.0
60-69	11.3
70-79	9.8

Thus, only one patient in nine fell below what is commonly considered normal blood calcium value.

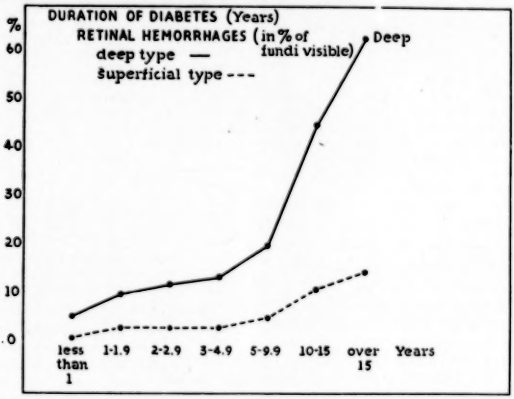

PLATE XVI.

basis of daily insulin dosage of 50 units or over, we find included ninety-two of the 2002 diabetics (4 1/2 per cent), and thirteen of the 730 deep hemorrhage eyes (1 3/4 per cent). From the standpoint of retinal hemorrhage in this diabetic group, our experience would indicate the only danger from insulin to be in withholding it. Plate XV.

X.—1.—g. *Deep Retinal Hemorrhages in Relation to Blood Calcium*

Reduced blood calcium has been advanced as a possible contributing factor in the production of retinal hemorrhages by Cammidge[31] and later by Lawrence[32]. Since Cammidge represented the normal blood calcium as 6 mg. per cent, he must have referred to the diffusible calcium. In this country, it is the feeling that more accurate methods exist for total blood calcium, which has for normal value a magnitude of 9-10 mg. per cent.

Nine of the deep retinal hemorrhage cases in

X.—1.—h. *Deep Retinal Hemorrhages in Relation to Blood Sugar*

Total Diabetics Bld. Sugar Recorded	Maximum Fasting B. S.	Deep Retinal Hemorrhages Right Eye	%
280	Under 140	38	13%
246	140-169	39	15%
242	170-199	51	21%
405	200-249	97	23%
242	250-299	59	24%
167	300-400	20	12%
24	Over 400	2	8%
1606		306	

The highest incidence of deep retinal hemorrhages does not fall in the low or in the high fasting blood sugar groups, but in the intermediate groups, as shown in the above table. Of the 306 deep retinal hemorrhages, right eye, only twenty-two (7 per cent) occurred in pa-

tients with a maximum fasting blood sugar over 300 mg. If deep retinal hemorrhages be plotted against fasting blood sugar over 300 mg. per 100 cc., there is no obvious correlation. Plate XV.

X.—2. *Waxy Exudates*

Waxy exudates in the retina occurred as follows:

WAXY EXUDATES

Age Group	Diabetics Fundi Visible	Diabetics Waxy Exudates	Diabetics %	Non-Diabetics %	Non-Diabetics Waxy Exudates	Non-Diabetics Fundi Visible
Under 10	130	0				
10-19	464	2	.4%			
20-29	274	4	1.4%	10.0%	2	20
30-39	315	1	.3%	.8%	1	129
40-49	512	42	8.2%	1.0%	3	305
50-59	1001	160	16.0%	.4%	1	229
60-69	957	165	17.2%		0	164
70 up	240	46	19.2%		0	54
	3893	420			7	901
Fundi not seen	108				not seen	13
	4001					914

If waxy exudates in diabetics are plotted according to the duration of diabetes, we obtain a curve with the same trend obtained in the deep retinal hemorrhage curve, except that it does not reach the same altitude, Plate XVII. The greater frequency of waxy exudates in the macular and perimacular area where deep hemorrhages are more numerous, the location of waxy exudates histologically, and the similarity of the incidence curves of deep hemorrhages and waxy exudates, would seem to indicate that waxy exudates may arise through hyalinization of deep retinal hemorrhages.

X.—3. *Nerve Fiber Layer Hemorrhages*

Nerve fiber layer hemorrhages have about the same incidence in diabetics (5 per cent) as in non-diabetics (4 per cent). In our series, nerve fiber layer hemorrhages were found in 196 of 3893 diabetic eyes, and in thirty-three of 901 non-diabetic eyes, in which the fundi were visible. Of the 196 nerve fiber layer hemorrhages in diabetics, thirty-four of them occurred alone, and 162 times they were found in conjunction with deep retinal hemorrhages.

NERVE FIBER LAYER HEMORRHAGES BY AGE DECADES

Age Group	Diabetics Fundi Visible	Diabetics Nerve Fiber Hemorrhages	Diabetics %	Non-Diabetics %	Non-Diabetics Nerve Fiber Hemorrhages	Non-Diabetics Fundi Visible
Under 10	130					
10-19	464	2	.4%			
20-29	274	6	2.1%	10.0%	2	20
30-39	315	7	2.2%	8.5%	11	129
40-49	512	19	3.7%	3.6%	11	305
50-59	1001	56	5.6%	2.6%	6	229
60-69	957	80	8.4%	.6%	1	164
70 up	240	26	10.8%	3.7%	2	54
	3893	196			33	901

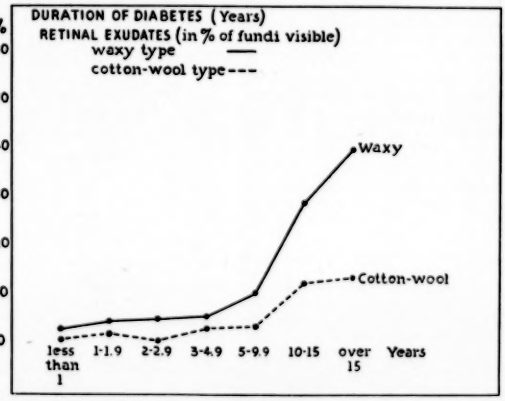

PLATE XVII.

Analysis of the clinical and blood chemical findings reveals no conspicuous abnormality in association with nerve fiber layer hemorrhages. The high incidence in non-diabetics under forty years of age is to be accounted for in the fact

that all hemorrhages listed occurred in cases of hypertension, nephritis, rheumatic heart disease, or hyperparathyroidism.

X.—4. *Cotton Wool Exudates*

Cotton wool exudates were found with the following frequency:

COTTON WOOL EXUDATES BY AGE

Age Group	Diabetics Fundi Visible	Diabetics C-W Exudates	Diabetics %	Non-Diabetics %	Non-Diabetics C-W Exudates	Non-Diabetics Fundi Visible
Under 10	130	0				
10-19	464	1	.2%			
20-29	274	3	1.1%	10.0%	2	20
30-39	315	5	1.5%	7.0%	9	129
40-49	512	19	3.7%	5.5%	17	305
50-59	1001	50	5.0%	2.6%	6	229
60-69	957	71	7.4%	.6%	1	164
70 up	240	19	7.9%		0	54
	3893	168			35	901
Fundi not seen	108				not seen	13
	4001					914

Cotton wool exudates show a rising incidence in the diabetics of our series, rising from zero to nearly eight per cent progressively through the decades of age, and a falling incidence in the non-diabetics, falling from ten per cent in the third decade to zero in the eighth decade. Of the 168 eyes of diabetics showing cotton wool exudates, seventy patients had NPN recorded as follows:

NPN	Cotton Wool Exudates
Under 35	34
35-39	22
40-44	8
45-50	5
Over 50	1
	70

X.—5. *Miscellaneous Fundus Abnormalities*

	Diabetics 3915 Visible	Non-Diabetics 901 Visible
Drusen in fundus	386	138
Degenerations	295	81
Conus	151	26
Iridescent crystals	28	4
Atrophy optic nerve	27	4
Proliferated capillaries in retina	26	0
Detached retina	16	1
Striate retinitis	12	5
Cupped disc	8	3
Opaque nerve fibers	7	1

Miscellaneous fundus abnormalities in diabetics and non-diabetics are listed here for record. Except for the proliferated vessels in the retina, and detachment of the retina consistent with retinitis proliferans, none of these miscellaneous abnormalities bear direct relationship to diabetes.

Striate retinitis may be a misnomer, for it refers to the deep striae seen best with scatter light, and located beneath the retina in the level of the retinal pigment epithelium. The oblique direction taken by the striae would suggest as a possible cause the traction lines resulting from the pull of the superior and inferior oblique muscles.

XI. The Optic Tracts

ABNORMALITIES OF THE OPTIC TRACTS

	4001 Eyes Diabetics	914 Eyes Non-Diab.
Sector defects*	48	
Homonymous hemianopsia*	24	
Toxic amblyopia	14	

*Fields recorded 3782 eyes of diabetics.

The maximum visual acuity with correcting lenses was recorded in 3980 of the 4001 eyes of diabetics. Unilateral anophthalmos was present in three diabetics. The corrected visual acuity was as follows:

	Rt. Eye	Lt. Eye
6/6 or better	1506	1503
6/7 and 6/9	247	240
6/12 and 6/20	135	134
6/30 and 6/60	41	49
less than 6/60	59	66
unrecorded	11	10
anophthalmos	3	
	2002	2002

In 3782 of the eyes of diabetics visual fields were recorded with the aid of perimeter and tangent screen. Sector defects were found in forty-eight eyes, and attributed to blocking of one of the main retinal vessels, to local detachments of the retina, and to glaucoma.

Homonymous hemianopsia was found in twelve diabetics (twenty-four eyes) and was attributed to hemorrhage, vascular block, or tumor behind the chiasm.

d. *Toxic Amblyopia* was found in fourteen eyes of seven of the diabetic patients of this series, all of the affected patients being males, all past the fifty-second year of age, and all showing sclerosis of retinal vessels of grade No. 2 or higher. The corrected visual acuity of the affected eyes ranged from 6/12 to 1/60 at the onset, and the visual field of each eye exhibited scotomata connecting with the blind

spot of Mariotte, largest for red and green, and presenting smaller dense cores tangent to the fixation point, consistent with the perimetric characteristics pointed out by de Schweinitz and Fewell[33], Doyne[34], Sattler[35], and Traquair[36]. We agree with de Schweinitz and Fewell in their contention that diabetics are susceptible to tobacco amblyopia, and we believe that improvement in visual acuity will follow abstinence from tobacco-alcohol unless the optic nerve or ganglion cells of the retina have already undergone degeneration.

It has been impossible for us to demonstrate scotomata, central or paracentral, of any type distinctive for diabetic processes.

XII. Ocular Muscles

Among 4001 eyes of diabetics, we found sixteen eyes with paresis or paralysis of one or more ocular muscles, the most common one being the external rectus. In only one of the sixteen cases did the fasting blood sugar exceed 300 mg. per 100 cc. The appearance of paresis or paralysis did not seem to bear any discreet relationship to the duration of the diabetes:

Duration Diabetes at Time Onset of Paresis-Paralysis	Number of Cases
Less than 1 year	3
1-1.9 yrs.	4
2-2.9 yrs.	5
3-4.9 yrs.	1
5-9.9 yrs.	3
	16

The sudden onset of the paralysis or paresis, and its slow disappearance, would suggest hemorrhage as a possible cause. Such a hemorrhage would have to be minute to involve but one ocular muscle.

XIII. Intraocular Pressure

Measurements by an accredited Schiötz tonometer were made in 3344 eyes of diabetics, with results depicted in block graph, Plate XVIII.

The distribution shown in this graph held for material sorted by age of patients, by duration of diabetes, by amount of insulin administered, by hypertension systolic and diastolic, and by abnormally high blood constituents.

Abnormally low ocular pressures were observed in coma cases provided the patient were seen at once before intravenous therapy was begun. This series includes 113 coma cases, of which eighty-five patients had coma once, and twenty-eight patients had coma two or more times. Negative pressure with a collapsed cornea was observed only once, namely in a sixty-five year old diabetic who did not survive coma. Usually, the ocular pressure rapidly resumed normal with replenishment of body fluids.

Abnormally high ocular pressures were observed with clinical evidence of glaucoma in twenty-one eyes:

Primary glaucoma	11 eyes
Secondary "	6 eyes
Hemorrhagic "	4 eyes

XIV. Summary and Conclusions

1. Diabetics as a group show a higher incidence of wrinkles in Descemet's membrane (Dm 26 per cent, non-Dm 10 per cent), deep retinal hemorrhages (Dm 18 per cent, non-Dm 3 per cent), depigmentation of iris epithelium (Dm 6 per cent, non-Dm 2 per cent), transitory refractive changes, and toxic amblyopia, than do non-diabetics.

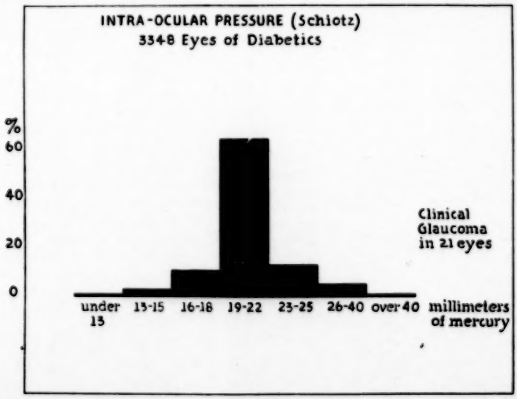

PLATE XVIII.

2. There is an equal incidence in diabetics and non-diabetics of cataracts of all sorts except flocculi in juvenile diabetics, of iritis, of atrophy of the optic nerve, and of arcus.
3. Wrinkles involving Descemet's membrane multiply with added age, but they show no obvious correlation with insulin administered, with blood sugar or NPN levels, or with ocular pressure.
4. Paresis of accommodation was demonstrated in twenty-one per cent of the diabetic patients so studied, and it was shown to be a transitory condition improving with diabetic therapy.
5. Glycogen infiltration of ocular tissues is a uniform accompaniment of diabetes.
6. Transitory refractive changes present in six per cent of the diabetics by history, and probably occur in a higher percentage of patients if the search be made with cycloplegia.
7. Senile and complicated cataracts occur with approximately equal frequency in diabetics and non-diabetics.
8. Slowly progressive, bilateral cataracts in juvenile diabetics were found eleven times in 297 patients. The morphological characteristics, fine flocculi and iridescent crystals in the cortical levels, with later changes in the posterior cortex at the pole, are indistinguishable from the cataracts of parathyroid tetany, scleroderma, or myotonic dystrophy.
9. Vitreous hemorrhage, associated with proliferation of connective tissue and blood vessels in retina and vitreous, is represented in a group of thirty-one diabetic patients, in whom the dominant findings are cardio-renal-vascular disease and disease of the retinal venous tree, and the prognosis for sight is poor in spite of the best diabetic handling.
10. Deep retinal hemorrhages in diabetics multiply with age, and multiply with continued duration of diabetes out of proportion to the age factor, but they show no obvious correlation with sclerosis of retinal vessels, with vascular hypertension, with renal disorders, with insulin dosage, or with blood sugar or calcium levels.

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CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

EDITED BY RICHARD C. CABOT, M.D.

CASE 21101

PRESENTATION OF CASE

First Admission: A thirty year old Canadian electric worker entered complaining of shortness of breath and cough of two months' duration.

Five months before entry he began to have many colds, noticed that he tired very easily and that he had some shortness of breath. At about the same time he had painless swelling in the left side of the neck which was not associated with sore throat and which subsided in about three weeks. He also noticed slight swelling of his face. About two months before entry the shortness of breath became worse and he developed a cough, more marked in the morning, with the production of about a teaspoonful to a quarter of a glass of greenish-yellow sputum without blood. He also had recurrent, sharp, stabbing, occasionally dull persistent pain in the left posterior chest, just medial to the scapula, which occasionally radiated to the right side. This pain was not worse upon coughing or deep breathing. He found that he could sleep better lying on his left side. These symptoms continued and about one month before entry he developed upper abdominal cramps severe enough to wake him at night. He also had a pressure feeling in his upper abdomen which was relieved by belching. He gradually became constipated and occasionally somewhat nauseated. During the past two weeks he had a swelling in the left anterior chest. His dyspnea became more marked and he was forced to use two pillows at night. There was no peripheral edema. He lost fifteen pounds during this illness. There had been no previous cough, pleurisy, chills or fever.

His family, marital and past histories are non-contributory.

Physical examination showed a well-developed and nourished man unable to lie flat in bed because of marked dyspnea. He had a harsh nonproductive cough. There were scratch marks over the chest and excoriations of the legs. His pharynx was red. There were two large, hard, movable, slightly tender supraclavicular glands three by two centimeters on the left. There were two smaller glands in the left axilla. In the region of the second and third ribs along the left sternal border was a diffuse, slightly tender, non-pulsating swelling four by three centimeters over which the skin was reddened. The left chest revealed dullness both anteriorly and posteriorly at the base with diminished voice and breath sounds, normal tactile fremitus and some medium moist râles. In the anterior axillary line at about the fifth interspace was an area of bronchial breathing, crepitant râles and egophony. No Grocco's triangle could be demonstrated. The neck and arm veins were distended while sitting. There was a large tortuous vein just to the left of the umbilicus, extending from xiphoid to pubis. The heart was not enlarged. One observer felt that the heart was slightly displaced to the right. The trachea was in the midline. The blood pressure was 140/86. The abdomen was distended, held tight, and was tympanitic. The right upper quadrant felt fuller than normal and was tender.

The temperature was 98.4°, the pulse 90. The respirations were 20.

Examination of the urine was negative. The blood showed a red cell count of 4,890,000, with a hemoglobin of 70 per cent. The white cell count was 9,000, 67 per cent polymorphonuclears. A smear was not remarkable. Two sputum examinations showed a few streptococci, a moderate amount of mucus, but no acid-fast bacilli. The stools were negative. A Hinton test was negative. The non-protein nitrogen of the blood was 46 milligrams. A tuberculin test 1:10,000 showed a very slight erythema at the end of forty-eight hours.

X-ray examination showed marked increase in the mediastinal shadow, due to irregular dullness, extending out to both hilus regions and obscuring the outlines of the heart. The borders of dullness were irregular in outline. There were also several patches of mottling in the periphery of the right lung field. The lower portion of the left lung field was obscured by homogeneous dullness which arose in the axillary line. The diaphragm on the right was low in position. The costophrenic angle was obliterated by a thin band of dullness which arose along the axillary line. The diaphragm on the right was low in position. The costophrenic angle was obliterated by a thin band of dullness which arose along the axilla.

On the fourth day a biopsy of an axillary gland was done. X-ray treatment was begun that day. Two days later a paracentesis of the left pleural cavity was done with the aspiration of 35 cubic centimeters of light yellow, slightly cloudy fluid which formed a very slight clot after standing. The fluid had a specific gravity of 1.020, with 7,500 white blood cells, 100 per cent lymphocytes and 2,200 red blood cells. The total protein was 4.3 per cent. A culture showed

no growth. The biopsy was reported as reticular hyperplasia.

On the fourteenth day he complained of considerable pain in his back. At this time he developed mild jaundice. He was discharged on the nineteenth day, following a course of x-ray treatment during which the swelling of his chest subsided.

Second Admission: Two weeks later.

For the first few days after discharge he felt somewhat improved, had more interest in things about him and craved sweets. His jaundice continued and itching of the skin became quite marked. During the ten days before entry he began to lose his appetite and strength. He had several periods of shortness of breath which were relieved somewhat by his physician's medication. During the two days before admission he had marked difficulty in swallowing and remained in bed.

He was admitted for further x-ray treatment. Examination showed a markedly emaciated, sick, jaundiced, drowsy man sitting propped up in bed. The sclerae were icteric, the skin showed many excoriations. There were hard, non-tender, enlarged glands in the left anterior and posterior cervical regions and in the left supraclavicular region. The left chest was flat anteriorly and posteriorly from the fifth rib down. In addition there were decreased tactile fremitus, breath and voice sounds and egophony. The abdomen was rigid and could not be palpated. The splenic dullness was increased. He died on the day of admission.

Differential Diagnosis

Dr. Walter Bauer: "He had painless swelling in the left side of his neck which was not associated with sore throat and which subsided in about three weeks." This might well have been swollen lymph nodes, or a tumor of the neck. I personally believe that it was swollen lymph nodes.

"He also noticed slight swelling of his face." It does not say whether it was unilateral or bilateral. I do not know that it makes much difference.

"About two months before entry the shortness of breath became worse and he developed a cough, raising about a teaspoonful to a quarter of a glass of greenish-yellow sputum without blood." I do not know which was the more frequent, a teaspoonful or a quarter of a glass of greenish yellow sputum. From that sentence alone it is difficult to say whether he had an irritative or a productive cough.

This pain was not increased by coughing or deep breathing, but he did find that he could sleep more comfortably on the left side. Therefore, I feel a little more inclined to interpret this pain as being pleural pain rather than due to some other cause.

"These symptoms continued and about one month before entry he developed upper abdominal cramps severe enough to wake him at night." In other words that was a real complaint because it did wake him out of a sound sleep.

"He gradually became constipated and occasionally somewhat nauseated." All I can say from the above is that something was going on within the abdomen as well as elsewhere, and whether that represented true intrinsic intestinal disease or not is a question.

"There had been no previous cough, pleurisy, chills or fever." In other words there was nothing to suggest that this was a flare-up of some previous chronic infection like tuberculosis.

From the history I think it is evident that we are dealing with a man who has upper mediastinal obstruction and in addition something in his abdomen. I think it is better for us to say that they probably are related. The only question is whether the abdominal symptoms are due to actual intrinsic intestinal disease or disease in the abdomen outside of the gastrointestinal tract.

"There were scratch marks over the chest and excoriations of the legs." Those, I think, are of significance. There is nothing said in the history about pruritus. We have evidence here that the man had a very definite pruritus as shown by the telltale marks found on physical examination; and these, with the previous history, should make us very suspicious of the most likely final diagnosis.

The chest signs, with the normal tactile fremitus and the other findings, we could interpret as being due to thickened pleura or partial collapse. In view of the previous history I should be inclined to think it means a thickened pleura.

"No Grocco's triangle could be demonstrated." In other words the physical examination is not consistent with a pleural effusion of any size. "The neck and arm veins were distended while sitting." Evidently he did have some interference with venous return when he assumed the sitting position.

In view of the laboratory findings we might question the amount and color of the sputum mentioned in the history. Nothing was found except a few streptococci.

"A tuberculin test 1:10,000 showed a very slight erythema at the end of forty-eight hours." I am unable to interpret this finding but I should be inclined to attach no significance to it.

From the history and physical examination I think it is obvious that we are dealing with a man who has a serious illness, an illness proceeding at a fairly rapid rate. He lost fifteen pounds in a very short period of time. We have evidence of some enlargement of lymph nodes.

We have evidence of mediastinal obstruction and abdominal symptoms. He gave no history of pruritus but we find evidence of it on physical examination. Therefore, without going any farther I think we should be quite suspicious that the thing we are dealing with is one process giving all the symptoms and signs, and it is probably a malignant lymphoma.

X-ray Interpretation

Dr. A. O. Hampton: He had two examinations, one before and one after x-ray treatment. This first one shows dullness at the left base which we interpreted as fluid. The right base shows fluid and there is this huge infiltrating mediastinal mass. There are numerous rather hazy areas of density scattered over both lungs. After treatment he shows definite improvement. The fluid at the left base has practically disappeared, but we have uncovered something in the region of that rib which may be in the lung, or disease of the rib.

Differential Diagnosis Continued

Dr. Bauer: We know that in the case of not only inguinal but also axillary lymph node biopsies the pathologist often returns a report of reticular hyperplasia or chronic inflammation. Therefore, if we can do a biopsy in some other region it is more likely to be of value.

I think that in this instance we are dealing with a man who has malignant lymphoma with widespread involvement. I think everything about the case is in keeping with this diagnosis. The only out is that this is the first time he has been exposed to x-ray therapy and although the mass on the chest wall diminished, he did not get such a satisfactory or lasting effect as we might expect. In view of his subsequently developing jaundice one wonders whether it was due to enlargement of lymph nodes with pressure on the bile duct or whether he had actual infiltration of the liver to such an extent as to cause jaundice. The fact that he had recurrent abdominal attacks could perfectly well be explained by an increase in size of intra-abdominal lymph nodes. I have no way of determining whether he did or did not have any actual involvement of the gastrointestinal tract wall. I think the other conditions that one would consider in the differential diagnosis would be those diseases in which one finds a generalized lymphadenopathy and an associated jaundice. I do not see that there is anything in the history or physical examination to point to such a diagnosis.

Dr. Hunter: Do we ever see itching marked enough to result in scratch marks in lymphosarcoma? I have never noticed it and I wonder if in this case it was not due to involvement of the kidney with resultant uremia.

Dr. Tracy B. Mallory: He was subicteric when he first came in and he also was suburemic. He has a double possibility.

Dr. William D. Smith: I remember this man very well. I saw him at the time of his first admission. I have nothing to add. We thought it was probably a lymphoma.

Clinical Diagnosis

Lymphoblastoma, ? type.

Dr. Walter Bauer's Diagnosis

Malignant lymphoma.

Anatomic Diagnoses

Carcinoma of the pancreas with extension into the duodenum and with metastases to mediastinum, lungs, bronchial, mesenteric and retroperitoneal glands, adrenals, kidneys, liver and heart.
Pleuritis, acute, with effusion.
Jaundice.
Early biliary cirrhosis.

Pathologic Discussion

Dr. Mallory: When this patient finally came to autopsy the difficulty I think was to find the patient in the face of all the tumor that he had. The largest single mass of tumor was in the mediastinum. It surrounded all the great vessels, the trachea, had infiltrated the pericardium, and passed across it into the heart itself, which showed a good-sized area of tumor invasion. The lungs were full of metastatic nodules. Both kidneys and both adrenals were completely replaced by tumor, virtually no kidney tissue was left and practically no adrenal. The liver was relatively uninvolved; it had only one tumor nodule. The spleen was absolutely negative. The mesenteric and retroperitoneal glands were all very large and the pancreas was totally replaced by tumor. Histologically it is a very undifferentiated small cell tumor, but I think it is definitely epithelial in character and I have very little hesitation in classifying it as a primary carcinoma of the pancreas.

CASE 21102

Presentation of Case

One month before entry the patient, a thirty-eight year old American housewife, first noticed the sudden onset of a non-productive cough. The cough was not accompanied by coryza, chest pain, or preceded by sinusitis. She gradually developed marked shortness of breath upon exertion but no edema or precordial pain. The cough, which was of a loud brassy quality, persisted steadily and was worse at night. She tried all kinds of medicines, including codein, without relief.

Her family and marital histories are noncontributory.

She had hay fever every summer during July and August, but no asthma. Eight years before entry she had an appendectomy and seven years before entry a thyroidectomy for colloid goiter.

Physical examination showed a well-developed and nourished woman lying propped up in bed coughing frequently and spasmodically but raising no sputum. There was a soft mass about five centimeters in diameter in the right side of the neck at the end of an old thyroidectomy scar. The vocal cords were negative. There were diminished breath sounds and tactile fremitus at both apices and scattered râles throughout the rest of the chest. The heart was negative. The blood pressure was 120/60. Abdominal examination was negative. There were no enlarged peripheral glands.

The temperature was 98.6°, the pulse 95. The respirations were 23.

Examination of the urine was negative. The blood showed a red cell count of 5,100,000, with a hemoglobin of 90 per cent. The white cell count was 10,500, 58 per cent polymorphonuclears.

X-ray films of the chest brought in by the patient showed a large mass anterior to the trachea and extending from the episternal notch down to the level of the bifurcation of the trachea. The trachea was markedly displaced posteriorly and was reduced to about one-third its normal diameter. The mass bulged more to the left than to the right, and its margins were fairly smooth. There was no evidence of cardiac enlargement or dilatation of the aorta.

Discussion

Dr. Oliver Cope: The account of the x-ray films brought in by the patient so narrows the diagnosis down that I shall do nothing but read the history, pass on to the x-rays and give the discussion afterward.

X-ray Interpretation

Dr. George W. Holmes: This film shows a definite enlargement of the mediastinal shadow with displacement of the trachea to the right. The smaller films show the trachea a little better and confirm the compression and displacement of the trachea.

Differential Diagnosis

Dr. Cope: It is evident that we are dealing with a mass in the mediastinum and I think everything else in the history and the physical findings can be explained on that basis. The problem therefore is to determine as far as possible from the history, the physical findings and the laboratory data what the nature of that tumor is. We have an unproductive cough, which is in favor of tumor. She had dyspnea on exertion, with no precordial pain. There is no mention of palpitation in the history, therefore I take it it was absent. From the fact that the man who wrote the history mentions no precordial pain I suspect that he was trying to limit it to some other organ than the heart. That might not necessarily be true, of course. Also, conversely, a tumor might involve part of a nerve root, giving an anginal-like pain. There was no relief from the cough. I should think that was of considerable significance. The fact that it was worse at night I do not believe is of much significance. It does not say that the patient could not lie down. The physical examination says that the patient was propped up in bed, coughing none the less. I do not think these things are of much help. The chest signs, dullness, diminished breath sounds and râles throughout the rest of the chest I should think were consistent with a large tumor in the upper mediastinum. I do not think they alter the diagnosis or are of any significance in the differential diagnosis.

The blood counts show an increase of red cells for a woman. The hemoglobin of ninety per cent I suppose is Tallqvist. It is higher than normal. I suspect that had an oxygen tension been taken we would have found it low. The red cell and hemoglobin increase are an attempt on the part of the blood to raise its oxygen carrying capacity, which commonly occurs with tracheal obstruction. I do not believe we need to suppose it is due to dehydration or any other thing.

The question now comes as to what might be the nature of that tumor compressing the trachea. It is stated in the history that there is sudden onset of coughing. I wish we knew a little more about the details of the suddenness of that onset. If it had been truly sudden it might be that we are dealing with a hemorrhage into a tumor, otherwise I do not explain it. Sudden onset is suggestive of a sudden increase in size in the tumor. The two things that might do it are hemorrhage and infection. And the history tries to tell us that there was no infection present at the time. The temperature is normal. No history of sinusitis or a coryza that would give us a lead to say thyroiditis or abscess rising perhaps from the trachea or esophagus; esophageal perforation or something of that sort.

There is no mention of a difficulty in swallowing and of course that is of primary importance in the diagnosis of a tumor in this position. I take it there was no difficulty in swallowing and therefore I shall have to say that it is unlikely that the esophagus was involved, either by a primary tumor or by extension. Of course the x-ray tells us that the tumor is anterior, with the trachea displaced posteriorly, but

that can happen in carcinomas arising in the esophagus, the larger bulk of the mass growing anteriorly and growing around the trachea, with tracheal compression. The absence of any difficulty in swallowing makes me omit the esophagus and think of something which would arise anteriorly to the trachea.

There are two other things in the physical examination that are of great importance. There is a mass in the right side of the neck near the old thyroidectomy scar. The larger bulk of the mass in the mediastinum is in the left side. Even though a thyroidectomy had been done for colloid goiter seven years ago, the tumor might perfectly well have regrown or the removal may have been incomplete, and the fact that the mass in the neck and mediastinum are on the opposite sides might naturally lead one to suspect that the lower poles had been incompletely resected and regrown. The lower pole on the right side remained in the neck and the lower pole on the left side dropped down into the mediastinum as it increased in size, if it was not there at the time of the original thyroidectomy. I am leading toward the thought, of course, that the mass is thyroid. The presence of a mass five centimeters in diameter in the right side of the neck with the position and character which are described suggests thyroid. It is important that the neck mass is on the opposite side from the larger part of the mass in the chest. Notice also that the vocal cords are said to be normal. Sometimes we are deceived. One may have malignancy in the mediastinum or in the region of the recurrent nerves and not get paralysis, but as a general rule if there is no paralysis of the vocal cords with a mass in the region of the recurrent nerves it argues against malignancy.

There are, of course, other tumors of the mediastinum, but the x-ray examination has gone a long way toward eliminating them; for example, aneurysms. The aorta is described as being normal. I am not sure of bilateral tracheal compression but I should think it was present from the x-ray films. A groove around the trachea would be usual in an uncomplicated aneurysm. I mean an aneurysm without perforation. A leaking aneurysm should be considered in view of the suddenness of onset of the symptoms, in which case the mass would be a mass of semi-organized blood clot; but again I should be in favor of ruling that out from the x-ray examination and also from the presence of the tumor on the right side in the neck consistent with thyroid.

To sum up, I should say that we are dealing with a tumor in the mediastinum which may or may not show hemorrhage into it. It is probably a colloid goiter and is the result of an incomplete resection at the operation seven years ago with regrowth. It is probably not malignant.

Clinical Discussion

Dr. Tracy B. Mallory: Dr. Wallace, will you tell us what they found at operation?

Dr. Richard H. Wallace: Dr. Allen performed the operation under intratracheal gas and ether. The mass in the right side of the neck was a large nodular goiter involving the whole right lobe of the thyroid. It was not connected with the mediastinal mass. Most of the left lobe had been removed at the previous operation but at the lower pole there was a pedicle about the size of a lead pencil, mostly blood vessels, which connected with the mediastinal tumor. This was separated with some difficulty in a definite line of cleavage and the tumor was delivered without splitting the sternum. It was a nodular goiter.

Dr. Frank T. Hunter: The comment I have to make is about the blood count. You sometimes see people with cyanosis and dilatation of the veins down the arms that have a high red count if you take it from the finger. If you take it from the toe you get a normal figure. It is important to know where the blood was taken.

Dr. Oliver Cope's Diagnosis

Colloid goiter.

Pathologic Diagnoses

(Substernal goiter.)
Hyperinvolution of thyroid.

Pathologic Discussion

Dr. Mallory: The specimen removed from this patient consisted of a large, substernal colloid goiter, measuring 9 by 7 by 3 cm., with another mass of equal size and similar appearance from the right lobe of the thyroid itself. There was no significant amount of hemorrhage, though a few small cysts contained bloody fluid. Microscopically it shows the typical hyperinvolution of a benign colloid goiter.

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SECTION OF DERMATOLOGY AND SYPHILOLOGY

WHEN the writer of this editorial returned from his study years in Europe in the autumn of 1895 he was invited to join the Boston Dermatological Club and at its first meeting he found himself, a mere stripling in more ways than one, in the midst of a group of elders wise in their knowledge of the world and in their knowledge of cutaneous medicine. These men were Dr. James C. White, Dr. Edward Wigglesworth, Dr. Francis B. Greenough, Dr. Abner Post, Dr. James Sullivan Howe and Dr. John T. Bowen.

This interesting group of men practically constituted dermatology in Boston forty years ago. Now we find a flourishing society devoted wholly to the interests of dermatology and syphilology, meeting enthusiastically four times a year and composed of seventy-five men and women, two-thirds of whom live in or near Boston, the remainder scattered over the New England states.

Last spring a small number of these men, led by Dr. Harvey P. Towle, thought the time ripe for the further advancement of dermatological interests in Massachusetts and applied to the Councilors of the Massachusetts Medical Society for permission to create a new section and this request was granted.

The first meeting of this new body will occur this coming June and a committee of its sponsors has arranged the program. The Chairman, Dr. Charles J. White, will narrate the early history of dermatology in Boston; Dr. C. Morton Smith will record a remarkable instance of a seeming cure in a syphilitic rarity; Dr. Arthur M. Greenwood will bring our knowledge of ringworm and its allied diseases up to date. The remaining two places on the program have been purposely thrown open to competition which will be adjudged by the organization committee of the section and anyone desiring to enter the lists may do so with the proviso that he send his finished paper to the Secretary of the Section, Dr. William P. Boardman, 388 Marlborough Street, Boston, not later than May first.

Dr. Greenwood's paper upon a disease which affects such a large percentage of our citizens and which is occupying the time of so many investigators in various parts of the world should attract to this new section meeting a large number of listeners and, we hope, discussers.

SECURITY TO PHYSICIANS

THE Bill known as Senate 52, "An Act providing Security to Hospitals and Physicians in the Enforcement of Reasonable Charges for Treatment of Certain Personal Injury Cases", is still under consideration by the General Court. It provides a reasonable method for dealing with a situation in which physicians are involved, sometimes against their will, for they rarely refuse to render assistance in emergencies or cases of accident, and in which physicians cannot now obtain a just recompense for services rendered. What is asked is that if a person is injured, and if claim for damages is made and paid, the claims of the physician and the hospital for recompense shall receive just consideration. These claims are to be given a statutory status which they do not now possess.

Since similar legislation in other states is reported to be working well, it is important that the present bill become law without essential change. Therefore, until the bill meets final disposition, physicians should follow up their representations made so well at the public hearing before the Joint Judiciary Committee, by continuing to remind Senators and Representatives of the necessity of finding a just way of dealing with what has become for the physician an intolerable situation. Do not let the General Court forget the interest of the physicians.

PUBLIC HEALTH

THE *Annual Report of the Surgeon General of the Public Health Service of the United States* is always an interesting document. The one for 1934, covering a period of a year prior to July 1, 1934, is no exception. Dr. H. S. Cumming, the Surgeon General, points out that during the year 1933 the general health conditions throughout the world were good in spite of the unfavorable economic situation. The United States was no exception and, as a matter of fact, conditions were somewhat better in America than in many other places. There appeared to be no evidence that the general health of the people, as far as the death rate was concerned, had been lowered as the result of the depression. The sickness rate, however, in many places where the depression was most acutely felt, was above the average for previous years. The great decimators of men, such as cholera and yellow fever, did not appear in the United States; the tuberculosis death rate continued to decrease and in 1933 was five per cent below the previous minimum rate; the typhoid fever death rate, as well as that for diphtheria, was the lowest ever recorded by the Public Health Service. Such a record must be attributed, in part, to the vast work of the relief agencies and the absence of any widespread epidemic. One feels, however, that the efforts of the Public Health Service of the United States must have played a considerable rôle in producing these results.

There were, nevertheless, three important crises during the years 1933 and 1934, affecting the public health. The first was the outbreak of epidemic encephalitis in St. Louis, Missouri, and the surrounding territory. During the summer and fall 1100 cases were reported with about 200 deaths. The seasonal incidence of this disease was strikingly similar to that of poliomyelitis, as was also its tendency to increased incidence in the less densely populated portions of the metropolitan district. Unlike poliomyelitis, however, the people most affected were in the later age groups. The general mortality was about twenty per cent. The type of encephalitis differed from the older, better recognized disease principally in that there was practically complete absence of paralysis of the eye muscles and recovery, when it occurred, was not often followed by sequelae. Secondly, there was an outbreak of amebic dysentery in Chicago, during the Century of Progress Exposition. Visitors who stopped at Chicago hotels where the disease had appeared became ill after returning to their homes in all sections of the country, but no epidemic prevalence of the disease was reported outside of Chicago. It was estimated that nearly 700 cases originated from the Chicago source. This outbreak was on a large scale and it was explosive in quality. From the point of view of the epidemiologist it was one of the first outbreaks in which a well-defined mode of transmission could be established with reasonable certainty. The sources were two hotels across the street from each other, which had, in part, a common water supply. This fact pointed to contamination of the drinking water as the most probable source of infection. Thirdly, in May 1934, poliomyelitis appeared in California, beginning in Los Angeles County and later extending northward. In May and June there were 1500 cases. A further report of this epidemic may be expected in the Surgeon General's summary of 1935.

With economic conditions as they have been for the majority of the people in the United States, in the last few years, good health has been a great blessing. Had we been swept by epidemics of influenza or other terrifying diseases, it seems certain that the steadfast courage of our people would have been less evident. Organizations such as the United States Public Health Service, fighting a battle against epidemic disease, enable us to escape from many of the serious pests which might otherwise so easily arise or be imported into this country.

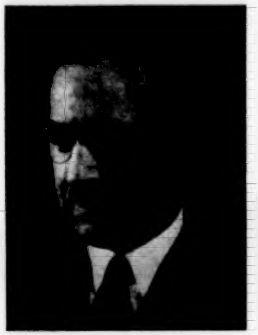
A STUDY OF ADEQUACY OF MEDICAL CARE

MEMBERS of the Massachusetts Medical Society have been asked, through a questionnaire, to give information of conditions existing throughout the state respecting the adequacy of medical service. This should meet with immediate response in order to supply information upon which action may be taken in formulating plans for devising methods of supplying the people with the services of doctors and nurses if such need is found to exist.

While there is evidence that the doctors are doing their utmost to provide good service for all in distress, and are, thereby, relieving municipalities of the burden of the cost of medical care, there is also evidence that the medical profession is in dire straits in many instances because of reduced incomes. It may be that the time has come when the burdens of free service must be lifted from the shoulders of practitioners. There are instances where incomes of doctors have fallen to a degree which is the cause of grave apprehension. How far this applies to Massachusetts should be ascertained. For New York City, for example, statements have been made that doctors have been obliged to take on unprofessional occupations in order to provide the necessities of life for their dependents, and a few instances of this situation have been reported in this Commonwealth.

The questionnaire should be filled out and forwarded immediately so that the true conditions may be tabulated and appropriate action taken.

A NEW DEAN AT HARVARD



DR. CHARLES SIDNEY BURWELL.

A GRADUATE of Harvard Medical School in the class of 1919, house officer at the Massachusetts General Hospital, teaching fellow at Harvard in 1921, instructor at the Johns Hopkins Medical School from 1921 to 1922, and associate in medicine until 1924; then, professor of medicine at Vanderbilt University until the present time —this is the record in medicine of the newly appointed Dean of the Medical School of Harvard University.

Dr. Charles Sidney Burwell is to be congratulated on his appointment to this, one of the most important medical posts in the country, and those who knew him here as a student and house officer and have followed his sound subsequent career, believe that the President and Fellows of Harvard University are also to be congratulated on having made a wise selection in filling the position so ably held by Dean Edsall.

Dr. Burwell's personality and social attributes have long endeared him to his many friends; his sound judgment and intellectual honesty have earned for him the respect of the scientific and medical worlds. In view of his scientific achievements, it is encouraging to believe that it is not the intention of the Harvard authorities to swamp him in a mass of administrative details, since his appointment as Dean of the Faculty of Medicine carries with it also an appointment as research professor of clinical medicine.

Harvard Medical School has won for itself a prominent position in the front rank of those institutions concerned with medical pedagogy and medical investigation. This position has been earned and maintained through the years by the wise selection of a medical faculty. Nothing, however, is permanent, and many changes are bound to come. It will be the constant care of the new dean to see that the foundations are strengthened, the fences kept in repair and the integrity of the institution preserved.

DR. MURK JANSEN

A CABLE just received announces the death on March first of Dr. Murk Jansen of Leiden, orthopaedic surgeon, *facile princeps* of Holland and President of the Société Internationale de Chirurgie Orthopédique. He visited America in 1922 and in 1934 and departed each time with the increased respect and affection of his American colleagues. He was a prolific writer of books and monographs, dealing scientifically with those factors which determine the normal and abnormal growth of bone. This is bold original work controverting previously accepted theories and propounding his own. Many developmental deformities he attributed to "Feebleness of Growth" often caused by too rapidly succeeding pregnancies and the consequent debility of the mother. The explanations which he offered were convincing to the degree of proof, and recent research has shown the soundness of one of his books in which he has challenged the reliability of the Meyer-Wolf law.

Jansen's character was a worthy companion of his intellect. His breadth of view, his openness of mind and his dignified gentleness left an even deeper impression upon those who knew him intimately than did his poly-lingual erudition. The Leiden orthopaedic field was lying fallow when he started his work nearly forty years ago. Almost alone he tilled it, meeting opposition rather than coöperation from the local medical hierarchy. The poor sought him at first and later all manner of men, as his fame grew and the miracles wrought by his wise brain and skilful hands became manifest. Half of his personal fortune went into the great hospital which is one of the heritages that he has left to his city and to his country. This tall, earnest gentleman, modest to the point of self-effacement and generous to the point of self-deprivation, was a fearless exponent of the truth as he knew it. It is fortunate that his peers grew to appreciate his greatness and to accept his leadership. There could have been no more fitting recognition of his preëminence than his election in 1933 to succeed Sir Robert Jones as President of the International Orthopaedic Society. He was essentially an internationalist; he spoke in many tongues; at the hearthstones of many lands he and his lovely wife found warm welcome.

R. B. O.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

PRATHER, GEORGE C. M.D. Harvard University Medical School 1924. F.A.C.S. Urologist, Newton Hospital. Assistant Urologist, Boston Lying-In Hospital. Assistant, Beth Israel Hospital. Consultant Urologist, Heywood Memorial Hospital. His subject is "Ectopic Testis as a Cause of Ureteral Dilatation. Case Report." Page 413. Address: 99 Commonwealth Avenue, Boston, Mass.

GRAVES, ROGER C. A.B., M.D. Syracuse University College of Medicine 1918. F.A.C.S. Urologist, Carney Hospital. Genito-Urinary Surgeon, Pondville Hospital, Wrentham. Member of the Associate Staff, New England Deaconess and Palmer Memorial Hospitals. Consulting Urologist to Quincy City Hospital, Winchester Hospital and Lakeville State Sanatorium. Genito-Urinary Consultant, Tumor Clinic of the Boston Dispensary. Associate Consulting Urologist, Brockton Hospital. Address: 12 Bay State Road, Boston, Mass. Associated with him is

MABREY, ROY E. A.B., M.D. Harvard University Medical School 1929. Formerly, Resident at Pondville Hospital. Address: 227 Beacon Street, Boston, Mass. Their subject is "Adenocarcinoma of Kidney Recurrent After Twenty Years." Page 416.

RILEY, AUGUSTUS. A.B., M.D. Harvard University Medical School 1907. F.A.C.S. Assistant Professor of Genito-Urinary Surgery, Harvard Medical School. Visiting Surgeon for Urology, Boston City Hospital. Consultant, Genito-Urinary Department, Boston Dispensary. Consultant in Urology, Malden Hospital. His subject is "Keratodermia Blennorrhagicum (Gonorrheal Dermatitis)." Page 417. Address: 868 Beacon Street, Boston, Mass.

KICKHAM, C. J. E. A.B., M.D. Harvard University Medical School 1927. Assistant Urologist, Pondville Cancer Hospital, Norfolk, and Carney Hospital, South Boston, Mass. His subject is "Calcified Hydrocele of the Tunica Vaginalis Testis. Case Report." Page 419. Address: 12 Bay State Road, Boston, Mass.

O'BRIEN, EDWARD J. M.D. Tufts College Medical School 1912. Visiting Surgeon, Cambridge City Hospital; Visiting Urologist, St. Elizabeth's Hospital; and Consulting Urologist, Somerville Hospital. His subject is "Report of Unusually Large Malignant Growth in Undescended Testis." Page 420. Address: 270 Commonwealth Avenue, Boston, Mass.

TROWBRIDGE, EDWARD H. A.B., M.D. Bowdoin Medical School 1884. F.A.C.S. Formerly, Visiting Surgeon to Worcester City Hospital. Surgeon-in-chief to the Harvard Private Hospital at Worcester, Mass. Past-President, Massachusetts Association Boards of Health. His subject is "Report of Urological Case Discovered in Course of Examination for Other Ailment." Page 421. Address: 36 Pleasant Street, Worcester, Mass.

MOORE, CARL R. B.S., M.A., Ph.D. Professor and Chairman of the Department of Zoölogy, University of Chicago. His subject is "Testicular Biology, Serotal Function and the Male Sex Hormone." Page 422. Address: University of Chicago, Chicago, Illinois.

WAITE, J. HERBERT and BEETHAM, WILLIAM P. See page 403, issue of February 28, for records of authors.

OPENINGS FOR YOUNG PHYSICIANS

FOR well-equipped young doctors, Maine offers attractive opportunities as indicated in the advertisement on page vi of this issue.

The people of this state are progressive, energetic and cordial. The medical association is well organized, and aggressive in its public health policies.

The invitation to settle in Maine should be considered by young physicians who are looking for openings.

Vol. 212
1935

The Massachusetts Medical Society

SECTION OF OBSTETRICS AND GYNECOLOGY*

THOMAS ALMY, M.D., *Chairman*, 140 Rock Street, Fall River, Mass.

C. J. KICKHAM, M.D., *Secretary*, 524 Commonwealth Avenue, Boston, Mass.

ARE EPISIOTOMY INCISIONS ADVISABLE DURING LABOR?

AN episiotomy properly done and repaired assures the patient of a perineum and vaginal orifice adequately supported and constricted as before delivery.

In the home, or where conditions are not ideal for surgery, an episiotomy should be done only where a laceration is imminent. In a well-equipped hospital a routine episiotomy can be performed especially where the accoucheur is using routine prophylactic forceps.

In the gradually dilating process of the slowly descending head it is impossible to determine before delivery the extent of the muscle separation as compared with muscle stretching. An episiotomy solves this problem and allows an anatomical reconstruction of the perineum.

Although there is much to be said in favor of the mediolateral episiotomy, I personally pre-

*A series of short selected articles by members of the Section will be published weekly.
Comments and questions by subscribers are solicited and will be discussed by members of the Section.

fer the midline episiotomy combined with a divergent incision when an anal sphincter tear is imminent. The repair of this type of episiotomy assures a symmetrical result because the incision separates the sphincter vaginae and the transversalis fascia in midline where a laceration usually occurs. It also follows the median raphe of the levator ani in the same line that the descending head usually separates the fibers.

The keynote of success in the anatomical repair of an episiotomy is the denudation of the mucous membrane from the underlying muscle layers and fascia. Failure to do this, that is, merely approximating the incised edges, often results in a subsequent rectocele, the reason for this being that the levator ani fibers have been separated by the descending head in the midline higher and deeper than the episiotomy incision has extended. The denudation process (best done by gauze dissection) clearly exposes all the muscle and fascia layers, and an accurate anatomical reconstruction is not only possible but simple.

Occasionally in cases where an episiotomy seems unnecessary and the child is delivered with no apparent laceration, careful palpation reveals an extensive submucous separation of the levator ani fibers in the midline. This means a rectocele later, and I believe one is justified in opening the perineum and approximating this muscle separation by a perineorrhaphy.

In the repair of any perineal laceration or episiotomy, I believe chromic 1 or 2 is adequate and satisfactory. I also strongly feel that silkworm gut sutures, as were formerly used, are not only a source of discomfort to the patient but a definite detriment to the healing process in many cases.

The Massachusetts Medical Society

LEGISLATIVE BILLS AFFECTING THE MEDICAL PROFESSION

Although reference has already been made to the following bills, the careful attention of the Fellows of the Massachusetts Medical Society is again directed to this list, now before the General Court and upon which final action has not yet been taken.

Bills which should be supported:

Senate 52, proposed by Senator Miles, was heard before the **Joint Judiciary Committee** on January 29. This bill contemplates the enforcement of reasonable charges for treatment by hospitals and physicians in certain personal injury cases. This bill was enthusiastically supported by the Massachusetts Medical Society and by various hospital representatives but needs further support from the individual members of the Legislature as well as from the members of the Committee.

House 756 is on the petition of the Massachusetts Medical Society and proposes to place in the hands of the Board of Registration in Medicine the power of approval of medical schools which train candidates for the licensing examinations. The Council of the Massachusetts Medical Society, at its October meeting, directed the Committee on Medical Education and Diplomas, the Committee on State and National Legislation and the Committee on Public Relations to confer and to propose suitable legislation designed to elevate the qualifications required for licensure in Massachusetts. As a result of these conferences this bill was introduced. It is an educational measure and is not aimed at any institutions but gives time for any school, which desires to do so, to raise its standards of training so that it can readily obtain the approval of the Board of Registration. This bill will be vigorously opposed, and already individual members of the Committee of the Legislature have been approached by the opposition. Every effort should be made to present the matter in its proper light to the members of the Legislature from your district. The hearing has been scheduled for March 7, before the **Committee on Education.**

Bills which should be opposed:

House 1157 is a bill to create a board of examination and registration to regulate the practice of chiropractic. This bill was scheduled to come before the **Public Health Committee** on March 7. It is evident that once more a very strenuous effort is being made to break down the present registration laws and it is hoped that the members of the profession will realize the serious nature of this attack and will make their influence felt in the proper direction.

House 1458 proposes to create a board of examination and registration to regulate the practice of magnetic healers. This bill has been assigned to the **Committee on State Administration.** It is another bill against the best interests of the public, and every effort should be made to oppose it. The hearing will be held March 8, at 10:00 A.M., in Room 423, State House, Boston.

Each Fellow is urgently requested to aid the Committee on State and National Legislation by appeals to each Legislator from his district.

MASSACHUSETTS LEGISLATIVE NOTES

H. 60. Bill requiring the vaccination of children in private schools. (Report, leave to withdraw. Accepted in House.)

H. 528. Bill providing for the regulation of practice of physicians and surgeons in certain cases. (Report, leave to withdraw. Accepted in House.)

H. 623. Petition of the Citizen's Committee Opposing Compulsory Vaccination for legislation to make vaccination and inoculation voluntary. Report, leave to withdraw. Accepted in Senate. (Final.)

H. 755. Petition of the Citizen's Committee Opposing Compulsory Vaccination that the public be protected from impure virus or serum and for a civil remedy for abuse thereof. Report, leave to withdraw. Accepted in Senate. (Final.)

MISCELLANY

A MEDICAL RACKET?

In connection with the prediction that under existing law, automobile insurance will be increased, Mr. Frank A. Goodwin, Registrar of Motor Vehicles, is quoted as declaring that "the insurance law in its present form has become a racket for doctors and lawyers".

We respectfully suggest that Mr. Goodwin should explain what he means by a "racket" for doctors, for we have the impression that the medical profession has not been fairly dealt with in many cases of automobile accidents.

THE RESIGNATION OF DR. HAMILTON

Dr. Alice Hamilton, Assistant Professor of Industrial Hygiene, at the Harvard Medical School, has tendered her resignation to take effect September 1, 1935.

CORRESPONDENCE

NEEDLESS REPETITION

February 23, 1935.

Editor, *New England Journal of Medicine,*

As one of the by-products of the depression, based upon an evident misconception of the meaning of a word, our language seems to have been enriched by a new expression which is being used by many doctors and frequently appearing in the columns of the *Journal*, i.e., "indigent poor". Any English dictionary will inform the inquirer that indigent means needy or being in want. Indigent poor, therefore, is another way of saying poor poor, when quite obviously it is the intent to designate those who are in needy circumstances and who are ill.

Yours truly,

H. F. R. WATTS, M.D.

6 Monadnock Street,
Boston, Mass.

RECENT DEATHS

BERGERON — FRANCOIS DEBORGIA BERGERON, M.D., of 120 William Street, Fall River, Massachusetts, died at his home, February 26, 1935. He was born in Montreal in 1870, and graduated from the University of Montreal Faculty of Medicine in 1894.

He is survived by four daughters, Mrs. Peter S. McMurray, Mrs. Altier Lavitne and the Misses Alice and Madeline Bergeron, all of Fall River; and five sons, Hector H., and Bernard L. Bergeron of Fall River, and D. Louis, Dominick A., and Paul Bergeron of Washington, D. C., and a brother, Hector J. Bergeron, of Montreal, Canada.

ST. JACQUES — ROBERT JOSEPH ST. JACQUES, M.D., of 71 Broad Street, Marlboro, Massachusetts, died in that city March 1, 1935. He was born in Canada in 1856 and graduated in medicine from the University of Montreal Faculty of Medicine in 1879. He practiced in Canada, New Hampshire, Whitinsville, and Spencer, Massachusetts, before settling in Marlboro. He retired two years ago.

Dr. St. Jacques is survived by two daughters, Miss Arline St. Jacques and Mrs. Wilfred J. Maybay, of Marlboro; a son, Emile St. Jacques, of Wareham, and a sister, Mrs. Christine Sarazen, of Three Rivers, Quebec.

JOSEPH—RAPHAEL N. JOSEPH, M.D., house physician at the Lakeville Sanatorium, died March 2, 1935. He was a graduate of the Georgetown University School of Medicine and had been an interne at St. Luke's Hospital, New Bedford.

He is survived by his parents, Mr. and Mrs. Nassar Joseph; a brother, Joseph Joseph, and a sister, Miss Mabel Joseph, all of New Bedford.

NOTICES

CLINIC AT THE PETER BENT BRIGHAM HOSPITAL

At 3:30 P.M. on Thursday, March 14, in the Amphitheatre of the Peter Bent Brigham Hospital, Dr. Henry A. Christian, Physician-in-Chief, Hersey Professor of the Theory and Practice of Physic in the Harvard Medical School, will give a medical clinic. To it are cordially invited practitioners and medical students. These clinics will be repeated on Thursdays until May.

On Saturdays in the wards of the Peter Bent Brigham Hospital, from 10 to 12, staff rounds will be conducted by Dr. Christian. These are open to all physicians.

LAWRENCE CANCER CLINIC

Established 1928

Lawrence, Mass., March 6, 1935.

To the Physicians of the North Half of Essex County:

Dear Doctor:

The regular Lawrence Cancer Clinic, to be held at Lawrence General Hospital, 1 Garden Street, Lawrence, upon Tuesday, March 19, at 10:00 A.M., will be a Demonstration Clinic with Channing C. Simmons, M.D., of Boston, Associate in Surgery at Harvard University Medical School, Surgeon-in-Chief to Collis P. Huntington Memorial Hospital, and member of the Cancer Commission of Harvard University, Boston, present as consultant. You are invited to accompany any of your patients whom you desire shall have this service, or to send them with a note, and a report will be returned to you. This service is gratis. Your attendance at the Clinic is always welcome.

This Clinic is endorsed by the Committee on Postgraduate Instruction of the Massachusetts Medical Society.

Committee

ROY V. BAKEMEL, M.D.,
CHAS. J. BURGESS, M.D.,
FRED'K D. MCALLISTER, M.D.,
JOHN J. MCARDLE, M.D.,
HARRY H. NEVERS, M.D.,
THOS. V. UNIAC, M.D.,
J. FORREST BURNHAM, M.D., *Chairman.*

REPORTS AND NOTICES OF MEETINGS

NEW ENGLAND ROENTGEN RAY SOCIETY

A meeting of the New England Roentgen Ray Society was held at the Boston Medical Library on the fifteenth of February. Dr. Healy presided and Dr. E. Ross Mintz, the speaker of the evening, gave a lecture on "Tumors of the Kidney." Dr. Mintz said that a complete understanding of the underlying pathology is a definite aid in the clinical as well as the x-ray diagnosis of kidney tumors. These tumors may be divided into two categories, first those of the cortex, including adenocarcinoma, hypernephroma, embryoma, and Wilms tumor; and secondly, tumors of the pelvis including epidermoid carcinoma and papilloma.

Adenocarcinoma and hypernephroma usually start as small nodules in the cortex. There is no change in the outline until the tumor encroaches on the pelvis or pushes the capsule outward. These neoplasms may reach enormous dimensions and may be of considerable size without showing any abnormality by x-ray. Hematuria is the usual sign after impingement on the pelves. As they grow they tend to invade the excretory portion of the kidney. Occasionally hypernephromas are accompanied by calculi, but it is the epidermoid carcinoma of the pelvis which is most commonly associated with stones. Tumors of long duration may lead to numerous small areas of calcification, and hypernephromas frequently invade the renal vein.

A case of leiomyosarcoma in a woman of sixty-five was demonstrated by means of lantern slides. The type of tumor cannot be distinguished by the pyelogram. Any adrenal tumor tends to push the kidney down, and can, therefore, be confused with other neoplasms. In only four per cent of cases do kidney tumors become necrotic, and, therefore, these patients very seldom run any temperature. It is uncommon to have the entire kidney tissue destroyed so that usually intravenous pyelograms show that some dye is excreted, while in hydronephrosis none is visible by x-ray. Most retroperitoneal tumors push the ureter over toward the midline.

Tumors of the anterior surface of the kidney are apt to produce hydronephrosis. It is often difficult to distinguish tumors of the pelvis from those arising in the cortex and secondarily invading the pelvis.

Benign papillomas arise in the kidney pelvis. Dr. Mintz mentioned several cases of this tumor causing hematuria in the prostatic age, and warned against blaming the prostate for hematuria in all cases in this age group. If such tumors are situated at the renopelvic junction, they are apt to obstruct the ureter, so that no dye will be excreted. It tends to invade the ureter and form papillomatous implants in the bladder wall, so that it is sometimes hard to tell whether the condition is primary in the bladder or in the kidney. These papillomas may likewise invade the secretory portion of the kidney. Epidermoid carcinoma of the pelvis is very malignant, tends to metastasize early and widely, involves the renal parenchyma, and tends to be associated with stones. The prognosis in such cases is rarely over five years and pain is the chief symptom.

Hypernephromas may cause a pulsating metastatic mass in the sternum. Embryomas usually occur in children and in eighty-five per cent of the cases the presenting symptom is a mass. For this reason the mortality is very high, only a dozen cures having been reported in the literature, and the condition usually being discovered so late that nothing can be done. Carcinomas of the kidney in children rarely metastasize to bone, although slides of such a case were shown. In the differential diagnosis nephritis may be confusing and a blood clot in the pelvis may cause definite changes in the pyelogram. It is important to remember that hemorrhage from tumors often starts after a slight trauma. Although we may say that generally in a solid tumor it is difficult to flare out the calyces in a retrograde pyelogram, and that this is less difficult in the cystic type of tumor, yet it is usually impossible to tell these two types apart.

A hemorrhagic cyst of the kidney may be confusing, and it is often difficult to distinguish infection from neoplasm by the x-ray, although the ureter is usually dilated in infection, and such patients have fever while fever is rare in tumors. It is advisable to order repeated pyelograms so that any changes that occur may be watched closely.

The end-results of fifty cases of renal tumors at the Massachusetts General Hospital were presented. Most of these cases came to the hospital with large tumors and with metastases in the chest. Forty-six per cent of the operated cases are still living, although some of them not long enough to be called cures. Thirty per cent of five year cures have been reported in the literature. The chances of cure are better if the patient reports the first indication of hematuria. Cases which are cured are practically always taken in the early stages.

In the discussion which followed, Dr. Sosman stressed the importance of the pathological understanding of a condition in order to properly read the x-ray. He said that Dr. Waters uses certain criteria for distinguishing hypernephromas from adenocarcinoma, the former causing an elongation and distortion without invasion of the kidney pelvis, while the adenocarcinoma both distorts and invades. However, the pathologist is often not able to distinguish these two types of tumor. Recently, good reports have come from Dr. Waters on the use of deep x-ray therapy before operating for these tumors. He presented several unusual cases from the Peter Bent Brigham Hospital. The first was a fifty year old woman complaining of pain in the flank with a bilateral distortion of the renal pelves and soft tissue masses. The second was a fifty-two year old woman with pain and presenting pyuria on the right, but showing by x-ray involvement of both sides, with a question of hydronephrosis on the right and a tumor on the left. Intravenous pyelograms showed poor function on both sides indicating that the tu-

mor may be a single benign cyst. Another case was shown where the left ureter was pushed far over to the left and the calyces greatly distorted. This was caused by a benign neurofibroma of the lumbar nerve.

Dr. Dresser said that metastases from hypernephromas sometimes pulsate and are clinically diagnosed as aneurysms. Dr. Mintz assured the audience that distortion was of no differential importance, and he questioned the value of the preoperative use of x-ray. Dr. Sosman said that Dr. Waters has been able to shrink a sizable kidney tumor, and in this way facilitated surgical removal. Films of a child's case were presented in which there was probably rupture of the upper calyx on the right side due to the use of too much dye.

MINERALS IN OUR BODIES AND OUR FOODS

Dr. Harry C. Trimble was the speaker on February 10, in the Course of Sunday Public Lectures at the Harvard Medical School. The subject was "Minerals in Our Bodies and Our Foods." The following are extracts from the presentation:

Minerals in the biochemical sense refer to the noncombustible residue or "ash" which remains after a tissue or a food is burned. In addition to those elements long included in this category, others less well known have been reported in recent years. These include fluorine, silicon, copper, manganese, and zinc, whose presence in minimum amounts seems to be definitely established; and aluminum, barium, boron, cobalt, nickel, lithium, rubidium, strontium, and titanium, whose presence have been reported by one or more observers. Refinement of and application of spectroscopic technique are revealing the presence of elements hitherto unsuspected. Whether all elements thus far listed as constituents of our bodies are present for a definite purpose, or whether a number may be merely the result of accidental lodgment of materials present in traces in the foods we eat, remains for future experimentation to determine.

The biochemical study of a particular element or substance contained in a food involves attempts to answer many questions. Among these are the following: (a) Is the substance present in a food? (b) In what amounts? (c) While in the gastrointestinal tract is its composition altered by enzymes, or by the simultaneous presence of other substances? (d) Is it soluble or insoluble in body fluids? (e) Is it excreted or absorbed? (f) What is its concentration in the blood? (g) Is the concentration contracted or does it fluctuate? (h) Does it pass into the tissues or is it excreted? (i) Is it utilized? (j) What are the degradation products? (k) Is an excess stored in the body? (l) What is the normal daily intake? (m) What is the effect of an excess or a deficiency of the substance?

In the body, water constitutes sixty-five per cent of the total weight, organic material twenty-nine per cent, mineral matter five per cent. The mineral ions obtained from digestion of foods are in general quite soluble. Calcium, phosphorus and iron may produce certain combinations which are of limited solubility and therefore present special problems. As the food is digested, the mineral ions are released into solution and, with some exceptions, the major percentage gradually passes into the blood. Some ions interfere with the solubility of others; for instance, one combination between calcium and phosphorus (tricalcium phosphate) which is formed in the middle and lower intestine is only slightly soluble. It passes through these regions largely unabsorbed. The fact that some calcium salts of other acids produced from normal foods also have a limited solubility was mentioned.

Extensive data of many investigators demonstrate a careful balance between acid and basic ions in the circulating blood. The concentration of basic ions fluctuates only very slightly in normal, and even the majority of abnormal, circumstances. Although the absolute quantities, and the ratios, of sodium, potassium, calcium and magnesium which enter the blood from time to time may vary over wide ranges, both the absolute amounts and the ratio of these substances to each other in 100 cc. of blood plasma remain very constant. The lack of uniformity in the distribution of mineral ions within the various separate parts of the body is well illustrated by the behavior of those chemically very similar ions, sodium and potassium. Sodium is in excess in the blood plasma and the extra-cellular fluids, while potassium is the more abundant within the red blood cells and muscle cells. Calcium and magnesium, likewise chloride and bicarbonate ions, have their own disparities of distribution among body tissues and between various body fluids.

The many possible dispositions of an inorganic ion, after it enters the body, are illustrated by calcium. This element may continue along the intestine and be excreted, or it may enter the blood. If it follows the latter course, it may be excreted when the blood passes through the kidney or it may pass into a gland and then enter a digestive juice and again return to the intestine, and perhaps reënter the blood once more. From the blood, calcium may enter the cells of muscles or other body tissues, or in the form of calcium salts it may be stored in the hard parts of the body, the bones and the teeth. From many of these locations it may again return to the blood at a later time. A state of physical and chemical equilibrium, undoubtedly influenced by other factors, exists between the calcium ions in the various fluids and parts of the body. If a disturbance in one locality occurs, the effect, theoretically at least, is felt throughout the organism and the disturbance may even influence the behavior of phosphate and still other ions.

The effects of a total deficiency of food upon mineral excretion have been shown in F. G. Benedict's classic experiment upon a fasting man. This revealed that the amount of sodium and chloride ions within the body is rigorously conserved when the intake of these is halted. From the second to the thirty-first day of fasting, the loss of sodium chloride

was reduced to only a small fraction of that occurring on the initial day. However, elements such as nitrogen, sulphur, phosphorus, calcium, magnesium, and potassium, that are contained within or associated with wasting tissues, tend to be excreted in quantities which diminish only to moderate extents during the fasting period.

If a person eats a wide variety of natural foods in reasonable quantities, there appears to be little danger that the intake of mineral elements will be deficient. In the case of calcium is it necessary that a special effort to include calcium rich foods should be made? Sherman, in his statistical study of many American diets, found that this element was consumed in amounts which provided a lower margin of safety for the individual than was true of other mineral elements.

BOSTON CITY HOSPITAL HOUSE OFFICERS' ASSOCIATION

A meeting of the House Officers of the Boston City Hospital was held on the evening of January 3 in the Thorndike Memorial Amphitheatre. Dr. Chester Keefer discussed "The Anatomical Diagnosis of Various Types of Chronic Arthritis." The diagnosis of this condition should be tridimensional; that is, etiological, anatomical, and functional, although this is not always possible. Joint diseases fall into two groups: the degenerative type and the proliferative type. In the first of these the primary disease is in the cartilage, while the other changes are secondary. The proliferative type of arthritis begins in the synovial membrane and the periarticular tissue.

Since the degenerative type is not an inflammatory condition, it should be called "arthrosis". Traumatic, hemophilic, and Charcot's joints are included in this type. Dr. Keefer showed a series of slides which demonstrated the gross and microscopic changes in these different types. As age increases the number of joints showing degenerative change also increases, the three most common being the knee, the acrominoclavicular joint, and the elbow. In the degenerative arthritis of the knee the medial facette in the patella is the first portion to show a degeneration of the cartilage. The damage varies from a slight erosion to complete eburnation. Histologically secondary changes are seen: first, fibrillation of the cartilage and a loss of elasticity; and secondly, a compression and thickening of the subchondral bone which attempts to fill in the cartilaginous defects resulting in a dense eburnated bone. Spurs are formed either by the flattening of the surface cartilage pushing the edges out, as in a mushroom, or occasionally new bone formation occurs at the edge of the joint.

In contrast to the above process, the proliferative type first shows a periarticular change without any cartilaginous pathology. There is an inflammatory reaction and infiltration of the synovial membrane which is normally only two or three cells in thickness. Polymorphonuclear cells are the first to infiltrate, but later there are lymphocytes and plasma cells. Following this articular surface damage, aggravated by movement, degenerative changes may occur. In rheumatoid arthritis, a particular form of proliferative arthritis, there is a synovitis involving the periarticular tissues which gives the typical fusiform joints. There is a projecting accumulation of fibrin, like heart vegetations, and later a proliferation and infiltration of the surface cells. When the joint becomes immobile because of the pain, the synovial membrane grows over the surface and forms fibrous adhesions and there is much atrophy in and above the joint, largely due to this immobilization. The cartilage degenerates and is replaced by new bone which in turn atrophies, leaving a thin irregular cancellous bone at the joint surface.

In the determination of the etiological factors in arthritis, cytological and bacteriological studies of the joint fluid are of distinct value in the proliferative type.

Dr. G. E. Haggart continued the discussion with a talk on "The Orthopedic Treatment of Chronic Arthritis." He showed slides of several cases of degenerative arthritis of the spine which usually occurs in squat, hard-working persons, and can usually be helped considerably by bed rest, diet, physiotherapy, and a back brace consisting of two uprights held snugly to the spine by straps. In some instances mechanical factors, such as lumbarization of the first sacral vertebra and a resultant unstable joint which is prone to strain, must be dealt with. Proliferative arthritis of the spine causes ankylosis of the joints with a practically solid column of bone. This occurs usually in young adults, and cannot be stopped once it has started, but deformities can be prevented by bed rest with hyperextension of the back to overcome muscular spasm, followed by a plaster jacket applied to hold the spine in the corrected position so that it will fuse in an advantageous position.

Dr. Haggart then spoke briefly on posture and its importance in arthritis. The long slender type of individual has poor muscles and poor posture and a constant ptosis. A corset for the young woman with this type of posture often is of value. Dr. Haggart believes that the individual should be trained to use his gluteal muscles which, when contracted, cause the pelvis to tilt backward, thus preventing a lumbar lordosis. The patient is first taught to contract these muscles while lying on his face. With gluteal muscle contraction, the legs also internally rotate and the feet invert. Pelvis rocking exercises while sitting in a chair are of value, and further exercises to keep the chest up and the chin in, help to give a proper posture.

The shoulder joint is not particularly subject to arthritis. In most instances pain in this region is due to subacromial bursitis, although cases of acromioclavicular arthritis are fairly frequent. The bursa may be injected with novocaine and the pain stopped for several days. Also the periarticular

structures may be injected in this way and use of the arm procured.

Foot strain leads to external rotation and to abduction and an abnormal weight-bearing line. In selected cases of chronic foot strain, manipulation under general anesthesia is employed to secure marked inversion and dorsiflexion followed by eversion with the object of breaking up adhesions. The forward part of the foot is then plantar flexed while the heads of the metatarsals are supported. In ambulatory cases a plaster boot is then applied with the foot inverted.

Hypertrophic arthritis of the hip, so often seen in elderly, obese patients who must be ambulatory, may be materially helped by an initial period of bed rest, followed by a walking caliper brace to remove the stress of weight-bearing on the joints. In selected cases arthroplasty or arthrodesis is employed.

Certain cases of the degenerative type of arthritis of the knee in obese women following the menopause are helped by thyroid. A flexion deformity of the knee in the rheumatoid type is very disabling and may be effectively treated by a wedging plaster or by the application of skeletal traction applied at the upper and lower tibia or os calcis. Traction is then exerted in the line of the deformity which is gradually corrected. Walking calipers are then applied.

The meeting was closed with an instructive motion picture on arthritis which was produced at the Mayo Clinic under the direction of Dr. Hench.

SEMI-ANNUAL MEETING OF MASSACHUSETTS TUBERCULOSIS LEAGUE

Attended by representatives of the twenty-eight Affiliated Organizations from most of the Counties of the State, the Semi-Annual Meeting of the Massachusetts Tuberculosis League was held at the Hotel Statler on February 7. In addition to the Massachusetts representatives there were guests from Maine, New Hampshire, Rhode Island and Connecticut.

Dr. Frederick T. Lord, President of the League, presided.

The speaker of the day was Philip P. Jacobs, Ph.D., Director of Publications and Extension of the National Tuberculosis Association. He took as his topic "A Challenge to the Future." Mr. Jacobs earnestly pleaded for a more intensive application of existing knowledge, an improved diagnostic service, especially in the cities, and a broader program of health education to reach all classes in the population. He stated that after almost thirty years' experience in the tuberculosis field he believed that the day when tuberculosis shall have ceased to be a serious problem can be hastened by at least ten years through an intensive drive by all health agencies in the community. He outlined in detail plans for such an intensive campaign. These included enlargement of the Boards of Tuberculosis Associations, Institutes for Public Health Nurses, an intensive Early Diagnosis Campaign, and an enlargement of the Sanatorium facilities throughout the State. He praised the Department of Public Health for its leadership in the tuberculosis field and urged the State League and its Affiliated Organizations to coöperate fully not only with the State Department but with the local Health Departments.

Following the luncheon meeting there was a meeting of the Board of Directors at which the Board endorsed Bills pending before the Legislature to permit the City of Cambridge to enter the Middlesex County Sanatorium District, and to permit the enlargement of Westfield State Sanatorium to care for 250 adult patients.

Present at the Directors' Meeting were the following: Dr. Frederick T. Lord, President; Dr. Francis P. Denny, Miss Josephine B. Colt, Rev. Walter F. Greenman, Mrs. Leslie B. Cutler, Professor Murray P. Horwood, Dr. Carl C. MacCorison, Mr. Raymond S. Patterson, Dr. Alton S. Pope, Miss Margaret Weir, Dr. John B. Hawes, 2nd; Mrs. H. G. Hamann, Mrs. A. L. Johnson, Mr. Clifton H. Hobson, Mrs. William C. Rogers, Dr. Samuel Hoberman and Mrs. Ralph S. Drury.

The Semi-Annual Meeting of the League was held during the week in which Mr. Jacobs conducted a Tuberculosis Institute for Tuberculosis Secretaries and Public Health Nurses. The Institute was successful far beyond expectation, the attendance always taxing the capacity of the meeting room. In addition to Massachusetts Secretaries and nurses there were representatives present from all the New England States except Vermont. Dr. Henry D. Chadwick, State Commissioner of Public Health; Dr. John B. Hawes, 2nd, President of the Boston Tuberculosis Association; Dr. Nahum R. Pillsbury, Superintendent of Norfolk County Hospital; Dr. Alton S. Pope, Director of the Division of Tuberculosis of the State Department of Public Health, and Professor Clair E. Turner were speakers on the Institute program.

MEDICAL CLINIC AT THE PETER BENT BRIGHAM HOSPITAL

Dr. Christian conducted one of the regular Thursday afternoon medical clinics at the Peter Bent Brigham Hospital on the seventh of February. He spoke briefly of a patient who thirty years ago developed a typical attack of coronary occlusion with agonizing precordial pain and prostration lasting six hours. Since this episode he had had recurrent attacks of anginal pain which recently had become more frequent until they would even occur while he was resting quietly. Last summer, however, after developing signs of decompensation, the attacks of angina stopped, and Dr. Christian pointed out that this is not infrequently the case in such patients. At postmortem the musculature of the cardiac apex was thinned and bulged out in an aneurysmal dilatation which in all likelihood represented the region of a previous infarct.

A patient was presented who one year ago en-

tered the hospital for ulcerations and inflammation of his legs. He had suffered with varicose veins for fifteen years and at the present time entered for treatment of another small ulcerated area. Four weeks before entry he developed some dyspnea on exertion, and physical examination showed a moderately enlarged heart, an enlarged liver, and edema. He was much improved after four days of bed rest. Dr. Christian discussed the condition of peripheral arteriosclerosis as shown in this patient. The radial artery was beaded and the brachial thickened and tortuous. This type of arteriosclerosis occurs usually as an aging process though it may occur prematurely; the vessel walls are apt to become calcified. There is no hypertension usually, and the blood flow is maintained without much pulse pressure. Sir Clifford Allbutt spoke of this type as decrescent arteriosclerosis to distinguish it from the form associated with hypertension.

This patient also showed some tremor of his hand and this suggests some arteriosclerosis in the cerebral vessels. Small and scattered skin lesions with silvery scales suggesting psoriasis were demonstrated above the varicose dermatitis. Dr. Christian contrasted the arteriosclerosis of this patient with the person who has hypertension but no change in his vessels until a later date. The first type is caused by a change in the vessel wall itself, while in the hypertensive type it may be due to the increased pressure, but this theory may be questioned because the increased pressure developed in the vessels of the upper extremities of people with coarctation of the aorta has been shown to cause no demonstrable change in the small peripheral arteries. In the hypertensive type the changes occur in the smaller vessels and may be due to some primary disease of these vessel walls, which is not clinically recognized and which causes the hypertension. Lewis believes that Raynaud's disease is caused by a condition which is primary in the vessel walls and acts to lower the threshold to such an extent as to allow nervous impulses to cause a spasm. It may be that in hypertension there is a similar disturbance of the arteriolar wall, and if this be true, the interruption of certain sympathetic nerves by the surgical procedures commonly in vogue at present are to be regarded with skepticism as to permanent benefit.

The patient also showed definite arcus senilis and Dr. Christian said that the common teaching that this condition is a manifestation of arteriosclerosis and old age is false, there being no connection with either; the true etiology not being known.

THE ANNUAL MEETING OF THE BOSTON TUBERCULOSIS ASSOCIATION

This meeting was held at the Sheltered Shop, 126 Newbury Street, Boston, February 25, 1935. In his annual report, Dr. John B. Hawes, 2d, president of the Association, expressed the feeling that Boston should contribute to the support of the Prendergast Preventorium, because of the benefit to the city in prevention of active cases of tuberculosis.

The Mayor of Boston, Mr. Mansfield, expressed sympathy with Dr. Hawes' attitude, but was doubtful of the right of the city to aid a technically private institution.

Other officers elected for the ensuing year are Dr. James J. Minot, vice-president; George S. Mumford, treasurer, and Mrs. Reginald Heber White, clerk.

THE ESSEX SOUTH DISTRICT MEDICAL SOCIETY

The Essex South District Medical Society held a regular meeting, and dinner, at the Addison Gilbert Hospital, Gloucester, on February 13, 1935, with the following program; Clinic, 5 P.M.:

1. Trichinosis. Dr. Earl Greene.
2. Unusual Gunshot Wounds. Dr. W. R. Irving.
3. Recurring Gummata of Skin. Dr. R. P. Hallett.
4. Intracranial Injury without Fracture of Skull. Dr. J. J. Egan, Jr.
5. Acute Leukemia. Dr. H. Carvell.
6. Pernicious Anemia with Cord Lesion. Dr. H. C. Burrell.
7. Fracture of Kidney with Slight Urinary Leak. Foreign Body in Stomach. Dr. S. W. Mooring.
8. Interesting Medicolegal Cases. Dr. I. B. Hull.
9. Use of Insulin in Nasal Cases. Meniere's Disease. Dr. T. H. Odeneal.
10. A Case of Traumatic Abdomen. Dr. W. W. Babson.
11. Bronchiectasis with Unusual Symptoms. Dr. G. S. Rust.

At the close of the dinner the society was entertained by a brief address by the Vice-President of the hospital corporation.

The President of the Society, Dr. Holtt, called attention to several pieces of legislation now pending, and urged attendance at the respective hearings.

The guest of the evening, Dr. Frank Pemberton of Boston, spoke on "Certain Aspects of Gynecology."

WM. T. HOPKINS, *Reporter.*

THE CHILDREN'S HOSPITAL MEETING

The annual meeting of the Children's Hospital, Boston, was held February 25, 1935, at the Old Colony Trust Company.

The board of managers reported that during 1934, 17,719 children visited the clinics 61,906 times. This hospital was started sixty-five years ago, and its purpose was to give to children of poor families expert medical care. The growth of the hospital has led to a broader policy, so that now all classes of children are accepted, and the institution has become a great teaching centre.

Samuel H. Wolcott was elected President; F. Murray Forbes, Vice-President; Edward L. Bigelow, Treasurer; F. Murray Forbes, Assistant Treasurer; James Garfield, Secretary, and Mrs. Samuel Eliot, Chairman of the Welfare Committee.

THE AMERICAN ASSOCIATION ON MENTAL DEFICIENCY

The Annual Meeting of the American Association on Mental Deficiency will be held at the Hotel Palmer, Chicago, on April 25, 26, and 27. The Thursday and Friday sessions will be devoted to studies on Mongolism, Birth Injury as an Etiological Factor in Mental Deficiency, Mental Disorders in Mental Deficiency, The Problem of Sterilization, Defective Delinquency and Its Relation to Penal Institutions, Community Supervision of the Paroled Mental Defective, and Newer Methods in Institutional Training for Community Life. The Saturday session, on April 27, will be devoted to the sociological, psychological, and the special educational aspects of Mental Deficiency. Physicians are cordially invited to attend these sessions. Complete data on the program may be obtained from the Secretary, Dr. Groves B. Smith, Godfrey, Illinois.

NEIL A. DAYTON, M.D., *Chairman*,
Committee on Public Relations,
American Association on Mental Deficiency.

MASSACHUSETTS MEMORIAL HOSPITALS

There will be a meeting of the Surgical Section in the Ladies' Aid Room (former nurses' dining room), Talbot Memorial, 82 East Concord Street, on Friday, March 8, 1935, at 12 noon.

Dr. William D. Rowland will talk on "Recent Advances in Ophthalmic Surgery" and there will be a discussion of Eye Pathology by Dr. Charles F. Branch.

MILO C. GREEN, *Secretary*.

CONVENTION OF THE CATHOLIC HOSPITAL ASSOCIATION

The Officers and Executive Board of the Catholic Hospital Association of the United States and Canada announce that on the invitation of the Very Reverend President and Board of Trustees of Creighton University, Omaha, Nebraska, the Twentieth Annual Convention is to be held at Creighton University, June 17 to 21, 1935, under the patronage of His Excellency, The Most Reverend Joseph Francis Rummel, D.D., Bishop of Omaha.

NEW ENGLAND PHYSICAL THERAPY SOCIETY

The regular meeting of the New England Physical Therapy Society will be held at the Evans Memorial Auditorium, 82 East Concord Street, Boston, at 8 P.M., on Wednesday, March 20, 1935.

The speaker of the evening will be Dr. L. L. Campbell, Professor of Physics, Simmons College, who will deliver the third in a series of lectures on Medical Electricity. The topics to be covered on the subject of "Light" are as follows:

Light: Visible and Invisible

The Nature of Light: Theories of, from Newton to Planck, and to-day. The Electromagnetic theory of light, the connecting link between Electricity and Light.

The Velocity of Light: The ether. Relativity.

Reflection and Refraction of Light: Laws of.

Polarization of Light: The Polariscope.

Diffraction and Interference of Light: The measurement of the wavelength of light. Units of wavelength.

Dispersion of Light: The spectrum. Color. Color-blindness.

Physicians and medical students are cordially invited to attend.

ARTHUR H. RING, M.D., *Secretary*.
Arlington.

THE SOUTH END MEDICAL CLUB

The next regular meeting of the South End Medical Club will be held at the headquarters of the Boston Tuberculosis Association, 554 Columbus Avenue, Boston, on Tuesday, March 19, 1935, at 12 noon. The speaker will be Frederick T. Lord, M.D., Member of the Board of Consultation, Massachusetts General Hospital; Clinical Professor of Medicine, Emeritus, Harvard Medical School. His subject will be "Lobar Pneumonia." All physicians are cordially invited to attend both lecture and luncheon.

THE NEW ENGLAND ROENTGEN RAY SOCIETY

ROUND TABLE CONFERENCE

The March meeting will be held Friday night, March 15, 1935, at the Boston Art Club, Boston, Mass., at 8 P.M.

Dinner will be served at 6:30 P.M.

SCIENTIFIC SESSION

Table No. 1. "Radiation Therapy." Dr. George W. Holmes.

Table No. 2. "Diseases of the Bones." Dr. P. F. Butler.

Table No. 3. "Pituitary Tumors: Diagnosis and Treatment." Dr. M. C. Sosman.

Table No. 4. "Chests." Dr. A. O. Hampton.

Table No. 5. "Gastro-Intestinal Tract." Dr. Richard Schatzski.

RICHARD DRESSER, M.D., *Secretary*.
695 Huntington Avenue,
Boston, Mass.

FOREST HILLS GENERAL HOSPITAL STAFF MEETING

There will be a meeting of the Forest Hills General Hospital Staff on the evening of March 11 at 8:00 at the Nurses' Home.

PROGRAM

Anesthesia. Dr. Sidney Wiggin.

Practical Anesthesia. Dr. Boris D. Rapoport.

WALTER S. DENNING, *Secretary of Staff*.

WORCESTER DISTRICT MEDICAL SOCIETY

The program for the next meeting of the Worcester District of the Massachusetts Medical Society is as follows:

Time: Wednesday evening, March 13.
Place: The Memorial Hospital, Belmont Street, Worcester, Mass.
Program: 6:15 P.M. (promptly) Buffet Supper.
7:15 P.M. Business Session.
7:45 P.M. Scientific Program.
(1) Postinfluenzal Pulmonary Complications. Dr. Oliver Stansfield.
(2) Round Table Discussion of Pediatric Prophylactic Measures. Drs. Charles Sparrow, Arthur Kimberly, Haskell Talamo and Constance Kaliris.
(3) Further Case Studies of Lumbo-Sacral Pathology as the Cause of Sciatica. Dr. Charles Ayers.
(4) Irradiation of the Ovaries in the Treatment of Carcinoma of the Breast. Dr. Richard Dresser.

ERWIN C. MILLER, M.D., *Secretary.*

PLYMOUTH DISTRICT MEDICAL SOCIETY

The next meeting of the Plymouth District Medical Society will be held at the Plymouth County Hospital, South Hanson, Thursday, March 21, 1935, at 11 A.M.

PROGRAM

Surgical Treatment of Pulmonary Tuberculosis.

1. Brief Summary of Surgery at the Plymouth County Hospital: Bradford H. Peirce, M.D., Supt.
2. Statistics: Roy F. Littlehale, M.D.
3. Pneumothorax: John McCarthy, M.D.
4. Case Reports: E. K. Jenkins, M.D.
5. Phrenic Operations, Pneumolysis and Thoracoplasty: G. A. Moore, M.D., Brockton.

Lantern slides and moving pictures.
Dinner at 1 P.M. G. A. MOORE, M.D., *Secretary.*

MIDDLESEX EAST DISTRICT MEDICAL SOCIETY

The next meeting of the Middlesex East District Medical Society will be held March 13 at the Middlesex County Sanatorium in Waltham, and will be in charge of Dr. Remick of that institution. Dinner will be served at one o'clock. Following the dinner there will be an inspection of the plant, and a discourse on the treatment of tuberculosis.

KENNETH L. MACLACHLAN, M.D., *Secretary.*

BOSTON CITY HOSPITAL—STAFF CLINICAL MEETING

A staff clinical meeting of the Boston City Hospital will be held Wednesday, March 20, 1935, at 8:15 P.M., in the Cheever Amphitheatre.

PROGRAM

Clinical Varieties of Neurosyphilis: Presentation of Cases. By Drs. H. H. Merritt and S. H. Epstein.
Pathological Findings in Neurosyphilis. By Dr. H. H. Merritt.
Treatment of Neurosyphilis. By Dr. S. H. Epstein.
Discussion: Dr. H. C. Solomon (by invitation) and Dr. W. P. Boardman.

COMMITTEE ON HOSPITAL CLINICS.

HARVARD MEDICAL SOCIETY

The next meeting of the Harvard Medical Society will be held in the Peter Bent Brigham Hospital Amphitheatre (Shattuck Street Entrance) Tuesday evening, March 12, at 8:15 P.M.

PROGRAM

Presentation of Cases.

A Trip Around the World by Airplane. By Richard U. Light, M.D., New Haven, Connecticut.

MARSHALL N. FULTON, M.D., *Secretary.*

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, MARCH 11, 1935

Tuesday, March 12—
1:30 P.M. Radio Program—WEEI. "Control of Typhoid Fever."
†2:30-4 P.M. Ward visit, Massachusetts Eye and Ear Infirmary.
†4-5 P.M. Seminar, Pediatric Laboratory, Massachusetts General Hospital.
4:30 P.M. Radio Program—WBZ. "Whooping Cough."
8:15 P.M. Harvard Medical Society. Peter Bent Brigham Hospital Amphitheatre (Shattuck Street entrance).

Wednesday, March 13—
All-day session. Greater Boston Medical Society Postgraduate Clinic Day at Beth Israel Hospital, Boston.

Thursday, March 14—
*12 M. Clinico-Pathological Conference. Massachusetts General Hospital.
†12 M. Clinico-Pathological Conference. Children's Hospital.
*3:30 P.M. Medical Clinic. Dr. Christian. Peter Bent Brigham Hospital.
†4:30 P.M. Surgical Clinic. Children's Hospital Amphitheatre.

Friday, March 15—
†12 M. Clinical Meeting of Children's Medical Staff, Massachusetts General Hospital. Ether Dome.
5 P.M. Radio Program—WEEI. "Prepare for Summer Vacations."
6:30 P.M. The New England Roentgen Ray Society. Dinner 6:30 P.M.; Meeting 8 P.M. Boston Art Club.

Saturday, March 16—
*10-12. Medical Staff Rounds. Dr. Christian. Peter Bent Brigham Hospital.

Sunday, March 17—
4 P.M. Harvard University. (Medical School, Building D, Longwood Avenue, Boston.) Free lecture. "Facts Regarding the Control of Diseases of the Gums." Dr. C. B. Vaughan.

*Open to the medical profession.
†Open to Fellows of the Massachusetts Medical Society.

March 7—Faulkner Hospital Clinical Meeting will be held at 5 P.M.

March 8—William Harvey Society will be held in the Auditorium of the Beth Israel Hospital, Boston, at 8 P.M.

March 8—Massachusetts Memorial Hospitals. See page 460.

March 11—Forest Hills General Hospital Staff Meeting. See page 460.

March 11, 12, 13—Surgeons to meet in Jacksonville, Florida. (Southeastern Surgical Congress). See page 83, issue of January 10.

March 12—Harvard Medical Society. See notice above.

MASSACHUSETTS DIETETIC ASSOCIATION

March 12—Tuesday, 8 P.M. "The Effect of Diet on Anemia," Dr. Lewis Diamond, Instructor in Medicine, Harvard University Medical School, Associate Physician, Children's Hospital.

March 19—Tuesday, 2 P.M. Field Trip: Visit Storehouse, First National Stores.

April 9—Tuesday, 8 P.M. "Small Hospital Problems," Miss Margaret Copeland, Superintendent, Free Hospital for Women.

March 13—Greater Boston Medical Society Postgraduate Clinic Day at Beth Israel Hospital. Symposium on Diabetes Mellitus, 9:30 A.M.-12:30 P.M. Luncheon, 12:30-1:30 P.M. Symposium on Biliary Tract Diseases, 1:30 P.M.-4:30 P.M. The Annual Dinner-Dance will be held at the Copley-Plaza Hotel. Apply to David B. Stearns, M.D., 465 Commonwealth Avenue, Boston, Mass., for particulars.

March 14—Clinic at the Peter Bent Brigham Hospital. See page 454.

March 15—The New England Roentgen Ray Society. See page 460.

March 19—Lawrence Cancer Clinic. See page 454.

March 19—The South End Medical Club. See page 460.

March 20—New England Physical Therapy Society. See page 460.

March 20—Boston City Hospital Staff Clinical Meeting. See page 461.

April 23—The Massachusetts Society for Social Hygiene will meet at the University Club, Boston. For information address Dr. E. Granville Crabtree, 99 Commonwealth Avenue, Boston.

April 25, 26, and 27—The American Association on Mental Deficiency. See page 460.

April 29 - May 3, 1935—The American College of Physicians will meet at Philadelphia. For information address Mr. E. R. Loveland, Executive Secretary, 133-135 South 36th Street, Philadelphia, Pa.

June, 1935—Medical Library Association will meet in Rochester, N. Y. For details, address the Secretary: Miss Frances N. A. Whitman, Librarian, Harvard University Schools of Medicine and Public Health, Boston, Mass.

June 17 to 21—Convention of the Catholic Hospital Association. See page 460.

June 27-29 inc.—British National Association for the Prevention of Tuberculosis will be held at Southport, England. Persons desiring further information should write to Miss F. Stickland, Secretary of the Association at Tavistock House North, Tavistock Square, London, W. C. I., England.

July 22-27—Seventh International Congress on Industrial Accidents and Diseases, Brussels, Belgium. The American Committee of the Congress is under the chairmanship of Dr. Fred H. Albee, New York, for the Section on Accidents, and that of Dr. Emery R. Hayhurst, Columbus, Ohio, for Industrial Diseases. The American delegation to the Congress will sail from New York on July 8 and visit London, Amsterdam, The Hague and Paris, and, optionally, Budapest. Physicians interested in the Congress or in the medical tour in conjunction with it, may address the Secretary, Dr. Richard Kovacs, 1100 Park Avenue, New York City.

DISTRICT MEDICAL SOCIETIES

ESSEX NCRTH DISTRICT MEDICAL SOCIETY

The Annual Meeting will be held in May. Time, place and subject to be announced.

E. S. BAGNALL, M.D., Secretary.

FRANKLIN DISTRICT MEDICAL SOCIETY

Meetings will be held on the second Tuesday of March and May at the Weldon Hotel, Greenfield, Mass.

CHARLES MOLINE, M.D., Secretary.

Sunderland.

MIDDLESEX EAST DISTRICT MEDICAL SOCIETY

March 13—See page 461.

May 8—Winchester.

K. L. MACLACHLAN, M.D., Secretary.

1 Bellevue Street, Melrose.

MIDDLESEX SOUTH DISTRICT MEDICAL SOCIETY

March 7—The meeting will be held at noon in the Hotel Continental, Cambridge, Mass.

NORFOLK DISTRICT MEDICAL SOCIETY

March 26—Fernald School for Feeble-Minded, Waverley. Details to be announced.

May—Annual Meeting. Date, time and place to be announced.

PLYMOUTH DISTRICT MEDICAL SOCIETY

March 21—See page 461.

April—Lakeville Sanatorium.

SUFFOLK DISTRICT MEDICAL SOCIETY

March 27—Clinical Meeting at the Boston Lying-in Hospital.

April 24—Clinical Meeting at the Children's Hospital.

The medical profession is cordially invited to attend these meetings.

ROBERT L. DeNORMANDIE, M.D., President.
GEORGE P. REYNOLDS, M.D., Secretary.

WORCESTER DISTRICT MEDICAL SOCIETY

March 13—See page 460.

April 10—Wednesday evening. Worcester Hahnemann Hospital, Worcester, Mass. 6:30 P.M.: Dinner. 7:30 P.M.: Scientific program and business session. Announcement of subjects and speakers to be presented at a later date. Dinner complimentary by the Hospital.

May 8—Wednesday afternoon and evening. Annual Meeting of the Worcester District Medical Society. The time and place of this meeting will be announced later.

ERWIN C. MILLER, M.D., Secretary.

27 Elm Street, Worcester.

BOOKS RECEIVED FOR REVIEW

Rats, Lice and History. Being a study in Biography, which, after twelve preliminary chapters indispensable for the preparation of the Lay Reader, deals with the life history of Typhus Fever. Hans Zinsser. 301 pp. Boston: Little, Brown and Company. $2.75.

A Textbook of Surgery. John Homans. Third Edition. 1231 pp. Springfield and Baltimore: Charles C Thomas. $8.00.

Good Food at Low Cost. R. T. Devereux. 38 pp. Pennsylvania: The Chester County Medical Society.

Stammering and Allied Disorders. C. S. Bluemel. 182 pp. New York: The Macmillan Company. $2.00.

Diabetes Mellitus and Obesity. Garfield G. Duncan. 215 pp. Philadelphia: Lea & Febiger. $2.75.

One Hundred and Fifty Years of Publishing. 1785-1935. 42 pp. Philadelphia: Lea & Febiger.

Jewish Contributions to Medicine in America. 1656-1934. With medical chronology, bibliography and sixty-nine illustrations. Solomon R. Kagan. 549 pp. Boston: Boston Medical Publishing Company. $5.00.

Diseases of Children. Edited by Hugh Thursfield and Donald Paterson. Third Edition. 1152 pp. Baltimore: William Wood & Company. $10.00.

A Textbook of Surgery. W. Wayne Babcock. Second Edition. 1312 pp. Philadelphia and London: W. B. Saunders Company. $10.00.

BOOK REVIEW

Amebiasis and Amebic Dysentery. Charles F. Craig. Illustrated. 315 pp. Springfield and Baltimore: Charles C Thomas. $5.00.

This excellent monograph, by one of the leading authorities on amebiasis and amebic dysentery, aims to provide the general practitioner and the health official with all of the available knowledge of the subject which he is likely to need. In addition, the work is profusely documented and a list of references appears at the end of each chapter, so that the student who may wish to consult original sources can do so without loss of time.

The reviewer heartily endorses Dr. Craig's book, is confident that it will be read widely, and offers his congratulations to the author.